Dissertation on

FACTORS INFLUENCING THE VISUAL OUTCOME OF OPTIC NEURITIS

Submitted in partial fulfillment of requirements of

M.S. OPHTHALMOLOGY

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THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2013

CERTIFICATE

This is to certify that this dissertation entitled **"Factors Influencing the Visual Outcome of Optic Neuritis"** is a bonafide record of the research work done by **Dr. E. RAJESWARI,** post graduate in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2010-2013.

Dr.K. NAMITHA BHUVANESWARI MS.,DO.
Chief
Squint, Neuro - Ophthalmology
and Paediatric Ophthalmology Dept.
RIO – GOH
Egmore, Chennai – 08

Dr.K.MARAGATHAM MS.,DO.
Director
RIO – GOH
Egmore, Chennai – 08

Dr. V. KANAGASABAI M.D.PhD
Dean,
Madras Medical College and Government General Hospital
Chennai – 03

Place:

Date:

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INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 04425363970

CERTIFICATE OF APPROVAL

To
Dr. E. Rajeswari
PG in MS Ophthalmology
Madras Medical College, Chennai -3.

Dear Dr. E. Rajeswari

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled " Factors influencing the visual outcome of optic neuritis " No. 07102011.

The following members of Ethics Committee were present in the meeting held on 20.10.2011 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan, MD	-- Chairperson
2.	Prof. A. Sundaram, MD Vice Principal , Madras Medical College, Chennai -3	-- Member Secretary
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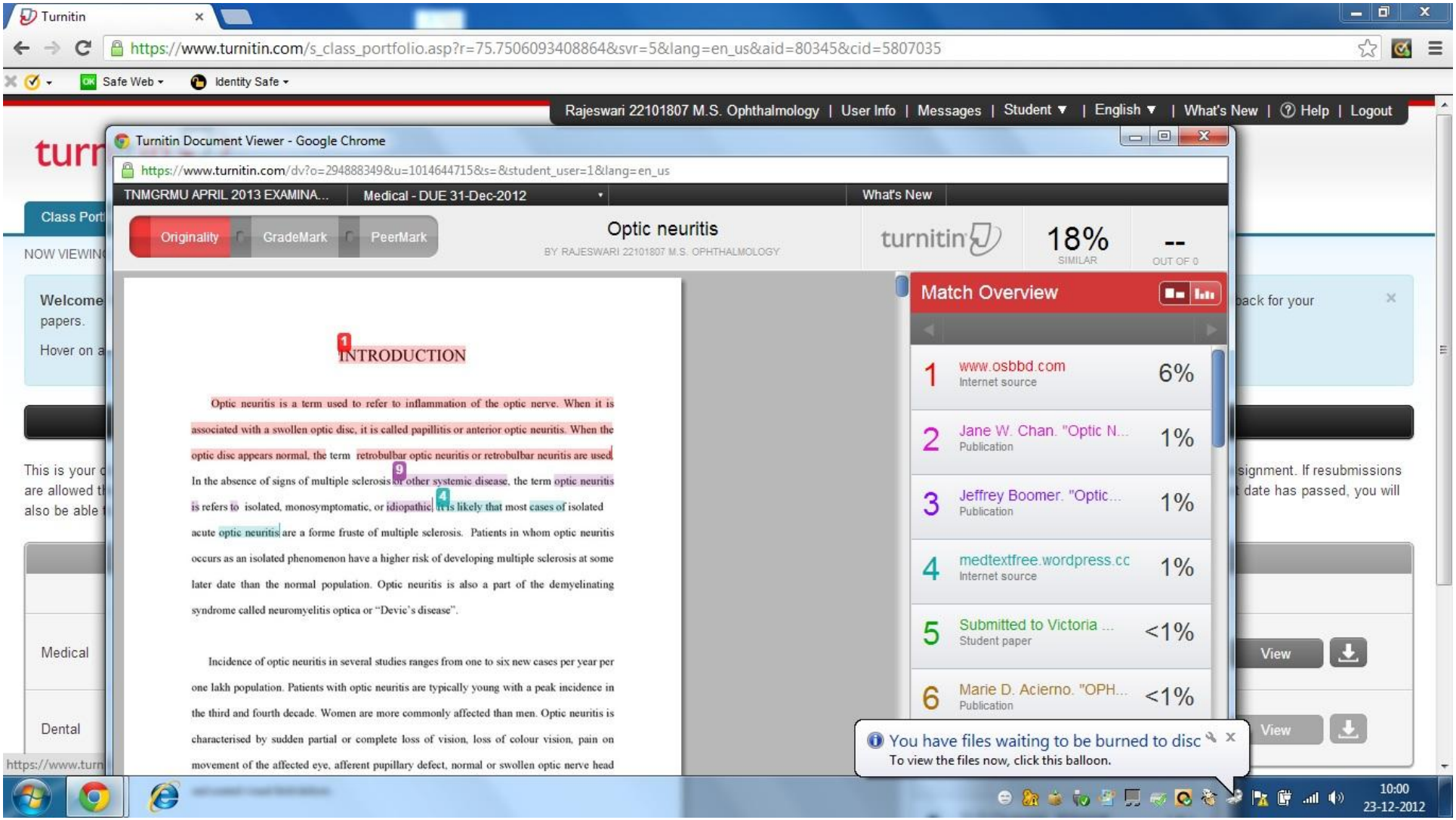
7.	Tmt. Arnold Soulina MA	-- Social Scientist
8.	Prof. Shanta Ravishankar Prof of Neuropathology, M M C, Chennai -3	-- Member

We approve the proposal to be conducted in its presented form

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee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INTRODUCTION
Optic neuritis is a term used to refer to inflammation of the optic nerve. When it is associated with a swollen optic disc, it is called papillitis or anterior optic neuritis. When the optic disc appears normal, the term retrobulbar optic neuritis or retrobulbar neuritis are used. In the absence of signs of multiple sclerosis or other systemic disease, the term optic neuritis is refers to isolated, monosymptomatic, or idiopathic. It is likely that most cases of isolated acute optic neuritis are a forme fruste of multiple sclerosis. Patients in whom optic neuritis occurs as an isolated phenomenon have a higher risk of developing multiple sclerosis at some later date than the normal population. Optic neuritis is also a part of the demyelinating syndrome called neuromyelitis optica or "Devic's disease".
Incidence of optic neuritis in several studies ranges from one to six new cases per year per one lakh population. Patients with optic neuritis are typically young with a peak incidence in the third and fourth decade. Women are more commonly affected than men. Optic neuritis is characterised by sudden partial or complete loss of vision, loss of colour vision, pain on movement of the affected eye, afferent pupillary defect, normal or swollen optic nerve head
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INTRODUCTION Optic neuritis is a term used to refer to inflammation of the optic nerve. When it is associated with a swollen optic disc, it is called papillitis or anterior optic neuritis. When the optic disc appears normal, the term retrobulbar optic neuritis or retrobulbar neuritis are used. In the absence of signs of multiple sclerosis or other systemic disease, the term optic neuritis is refers to isolated, monosymptomatic, or idiopathic. It is likely that most cases of isolated acute optic neuritis are a forme fruste of multiple sclerosis. Patients in whom optic neuritis occurs as an isolated phenomenon have a higher risk of developing multiple sclerosis at some later date than the...

ABBREVIATIONS

ONTT	Optic Neuritis Treatment Trial
CSF	Cerebrospinal fluid
MS	Multiple Sclerosis
HIV	Human Immunodeficiency virus
VEP	Visually Evoked Potential
AION	Anterior Ischemic Optic Neuropathy
RAPD	Relative Afferent Pupillary Defect
VDRL	Venereal Disease Research Laboratory
FFA	Fundus Fluorescein Angiography
MRI	Magnetic resonance imaging
HLA	Human leukocyte antigen
NMO	Neuromyelitis optica
CMV	Cytomegalovirus
HBV	Hepatitis B virus
EBV	Ebstein Barr virus

CONTENTS

INTRODUCTION

Optic neuritis is a term used to refer to inflammation of the optic nerve. When it is associated with a swollen optic disc, it is called papillitis or anterior optic neuritis[1]. When the optic disc appears normal, the term retrobulbar optic neuritis or retrobulbar neuritis are used. In the absence of signs of multiple sclerosis or other systemic disease, the term optic neuritis refers to isolated, monosymptomatic, or idiopathic. It is likely that most cases of isolated acute optic neuritis are a forme fruste of multiple sclerosis. Patients in whom optic neuritis occurs as an isolated phenomenon have a higher risk of developing multiple sclerosis at a later date than the normal population. Optic neuritis is also a part of the demyelinating syndrome called neuromyelitis optica or "Devic's disease".

Incidence of optic neuritis in several studies ranges from one to six new cases per year per one lakh population. Patients with optic neuritis are typically young with a peak incidence in the third and fourth decade. Women are more commonly affected than men. Optic neuritis is characterised by sudden partial or complete loss of vision, loss of colour vision, pain on movement of the affected eye, afferent pupillary defect, normal or swollen optic nerve head and central visual field defects.

Optic neuritis can spontaneously recover or can lead to permanent optic nerve damage leading to permanent visual loss. Spontaneous recovery can be speeded up by parenteral steroids (Optic Neuritis Treatment Trial) at appropriate time. Permanent optic nerve damage can be prevented by this.

REVIEW OF LITERATURE

Kirkham and Coupland – 1981 found that the most common findings after an attack of acute optic neuritis were optic atrophy, defective colour vision and prolonged pupil cycle time.

Griffith (1897) and Gunn (1904) considered the central field to be always affected in patients with optic neuritis.

Hierons and Lyle (1959) and Kennedy and Carroll (1960) found that optic neuritis occurring in children is more common in first and second decade of life.

Kahana et al (1976) found that multiple sclerosis developed in only 13% of patients with anterior optic neuritis, compared with 60% of patients with retrobulbar optic neuritis.

Bradley and Whitty (1967) believed that ultimate visual acuity after optic neuritis has no bearing on the subsequent development of multiple sclerosis.

Kurland et al (1966) Bradley and Whitty (1967) Morrissey et al (1995) concluded that most of the adult patients with bilateral simultaneous optic neuritis have the same risk of developing multiple sclerosis as patients with unilateral optic neuritis.

1988 – Optic Neuritis Treatment Trial - a cohort study of patients with acute unilateral optic neuritis followed up for 15 years to assess the prognosis of optic neuritis, the use of steroids to treat it and the subsequent risk of developing multiple sclerosis was started.

2006 – Final examination of patients enrolled in ONTT was done.

EMBRYOLOGY

OPTIC NERVE

Axons

The optic nerve develops from the embryonic optic stalk, which appears at the fourth week and connects the optic vesicle to the forebrain. As the stalk lengthens, it becomes thinner and the lumen is progressively occupied by the axons growing from the ganglion cells of the retina (the seventh week, 15-mm stage). In the meantime, the embryonic cleft closes at the sixth week of gestation. At the eighth week, axons fully occupy the stalk and reach the brain and a rudimentary optic chiasma is established. The mechanism by which the embryonic retinal ganglion cell axons reach the optic disc remains unclear. Many factors, such as the paired box containing the *Pax2* gene the axon guidance molecule netrin-1 and other cell surface or extracellular matrix component may be involved in axon path finding mechanisms. Expression errors of these molecules lead to optic nerve hypoplasia[2]. The axons of the optic nerve are surrounded by myelin sheaths. Myelinization begins centrally, progresses in a centrifugal direction toward the eye, and terminates at the level of the lamina cribrosa. The myelin sheath is produced by oligodendrocytes, and myelinization is usually complete shortly after birth.

Optic Nerve Sheaths

The sheaths of the optic nerve begin to appear at the end of the seventh week. Thin, elongated mesenchymal cells surround the optic nerve (10-mm stage) and become a single compact layer by the 17-mm stage. The pia mater can be

identified by the ninth to tenth week of gestation (45- to 50-mm stage), followed by the dura mater at the fifth month of gestation and the arachnoid sheath by the sixth and seventh months of gestation. Both the pia mater and the arachnoid are derived from the neural crest. **(Figure 2)**

Glial Element

At the ninth week (45-mm stage), the glial cells in the optic nerve are oriented in rows between the fascicles of axons. A peripheral layer of glial cells forms a glial limitans made up of immature astrocytes with glial filaments under the thin meningeal sheath. The glial limitans is separated from the pia mater by a complete basement membrane. Astrocytes line the connective tissue septa and capillaries. They are distributed among the axons at the 200 mm stage.

It has been suggested that optic nerve oligodendrocytes may originate from the astrocytes that occupy the optic nerve before myelinization rather than exclusively from glioblasts[2]. The glial cells in the optic nerve and retina may differentiate into astrocytes and oligodendrocytes.

It is not clear whether microglia originate from mesoderm or ectoderm, although most studies support a mesodermal origin. Under the electron microscope, microglia are identified in the optic nerve at the eighth week of gestation. Most microglia are found within bundles of axons, and there is no preferential distribution in relation to blood vessels or to the pial surface at any stage of development. The percentage of microglia present increases from 1.3% at 8 weeks to 2.7% at 18 weeks.

Vasculature

The development of capillaries in the optic nerve and the CNS is similar. At the 11th week (65mm to 73mm stage), vessels and connective tissue from the pia mater begin to enter the proximal optic nerve and slowly enlarge the connective tissue septa during the next few months. The capillaries within the optic nerve are separated from the axons by a relatively complete astrocyte sheet and perivascular space. In the 18th week (160mm stage), vascularization of the optic nerve is completed, and there may be anastomoses anteriorly by this stage with the arterial circle of Zinn-Haller. The capillaries within the neural tissue are surrounded by astrocytes and by a partially fused double basement membrane of both endothelial and glial cell origin. The basement membrane along the astrocytic foot processes defines the limits of the connective tissue septa along the neural tissue[2].

Figure 1: Visual pathway

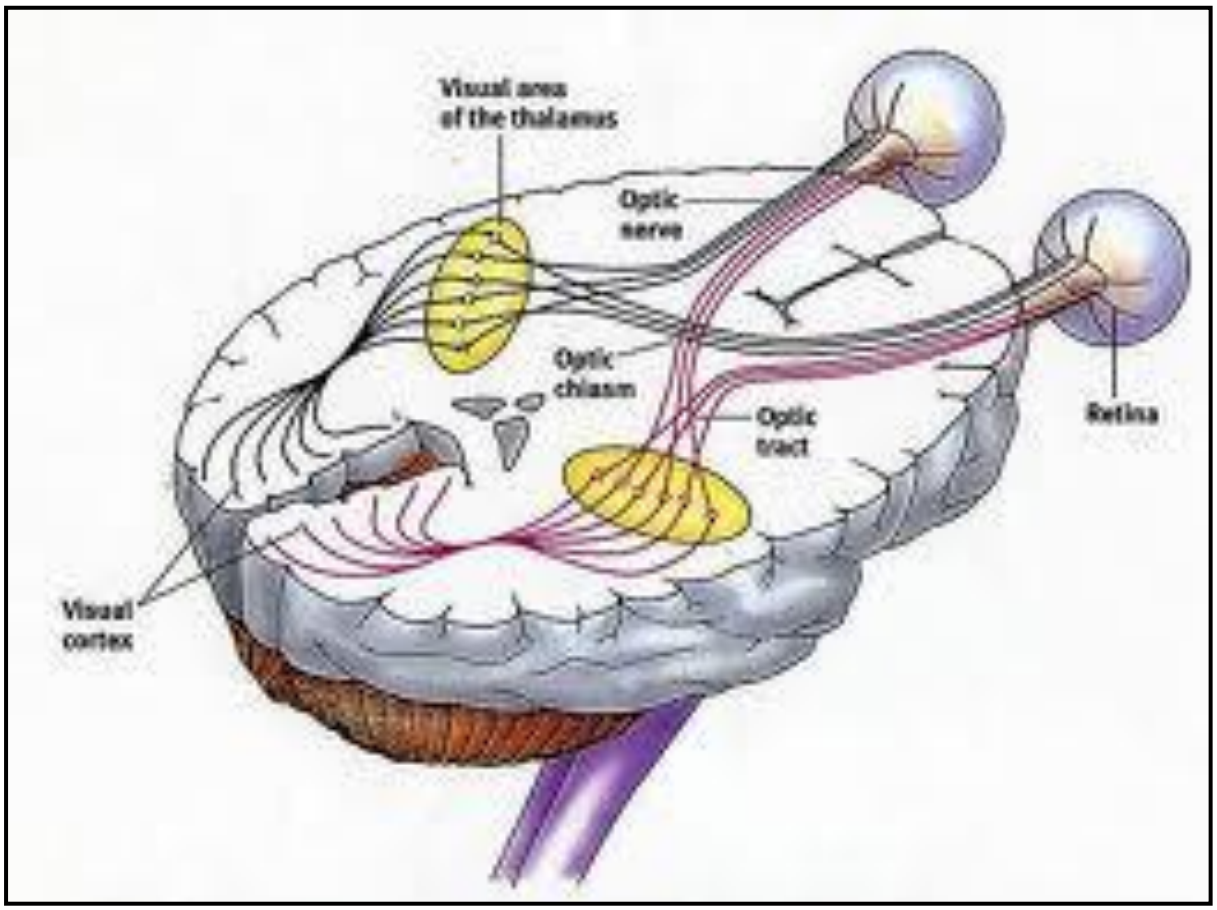
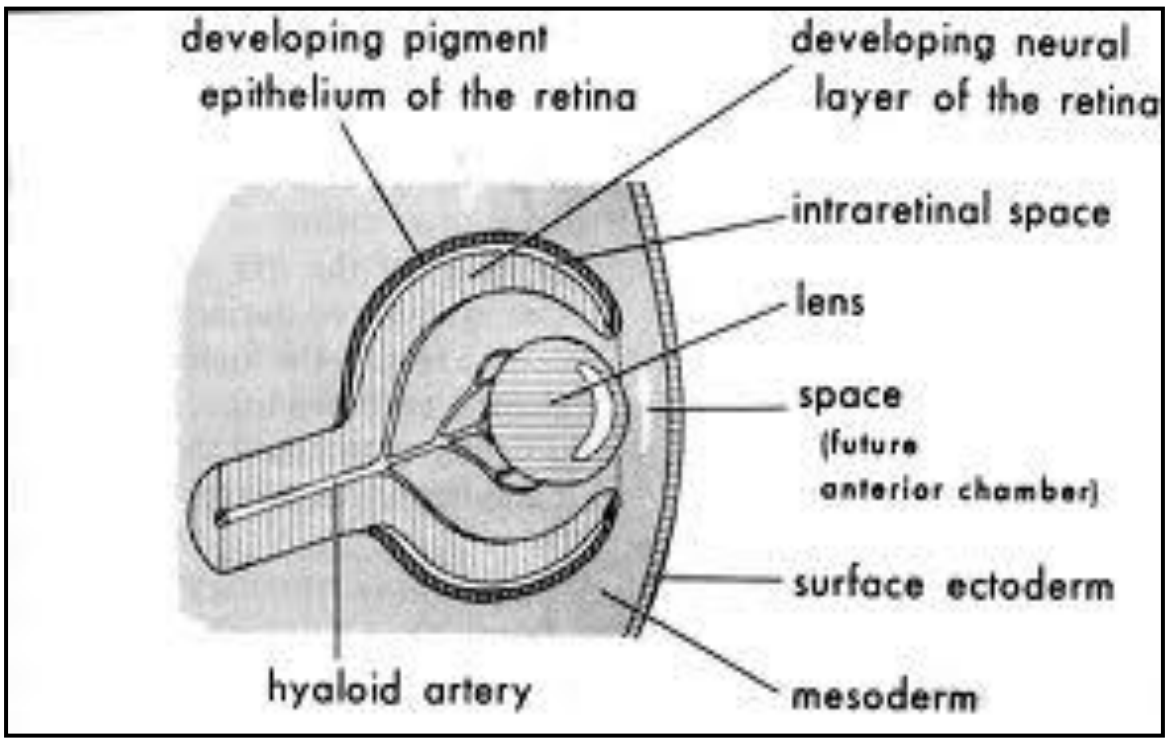
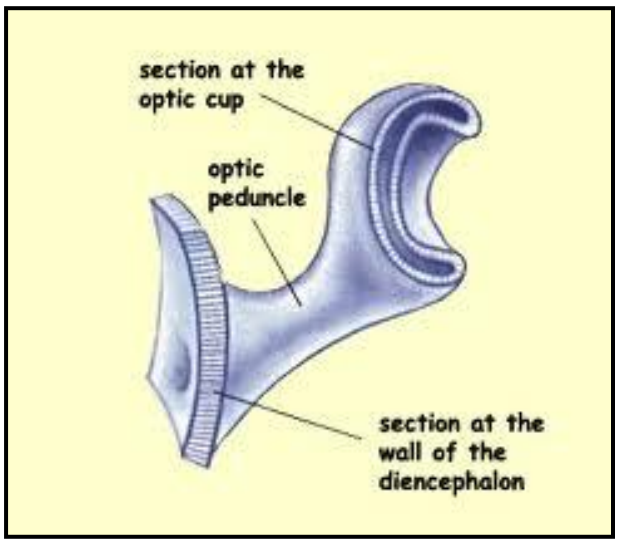


Figure 2: Development of optic nerve





ANATOMY OF THE OPTIC NERVE

The optic nerve is not really a nerve as are the peripheral nerves. It is a part of the central nervous system. As such it is a tract and its axons are myelinated by oligodendrocytes, not schwann cells. Optic nerve carries about 1.2 million retinal ganglion cell axons. The axons form the nerve fibre layer and eventually synapse in the lateral geniculate body. The optic nerve is about 50 mm long. It is divided into four sections – intraocular, intraorbital, intracanalicular, and intracranial[3]. These zones have very different microanatomic arrangement with elements of neuroectoderm and mesoderm intermingled. **(Figure 3-5)**

Intraocular Optic nerve (Optic nerve head):

Axons of the retinal ganglion cells form bundles that constitute the nerve fibre layer which then converges like spokes of a wheel at the optic nerve head. It is about one mm long. Its diameter is 1.5 mm horizontally by 1.8 mm vertically at the level of the retina. The intraocular optic nervc can be further divided into three anatomically distinct zones – retinal or prelaminar (anterior), the choroidal or laminar portion (middle), and the scleral or retrolaminar portion (posterior). Optic nerve includes an oval grouping of 200 to 300 holes that perforate the choroid and sclera forming a specialized structure the lamina cribrosa through which all retinal axons pass to exit the eye. The anterior surface of the nerve head is the clinically visible optic disc. The appearance of the disc depends upon two important features: size of the scleral canal and the angle of exit of the canal from the eye.

- Surface area is supplied by retinal capillaries.
- Prelaminar portion is supplied by the peripapillary choroidal vessels.

- The laminar portion of the optic nerve head receives its blood supply from circle of Zinn, formed by short ciliary vessels.
- The postlaminar area is supplied by branches of pial plexus from central retinal artery.

Intraorbital optic nerve:

The orbital portion of the optic nerve is 25 mm in length and thus exceeds the antero-posterior distance from the back of the globe to the optic foramen by about eight mm. The redundancy of the sinuous optic nerve permits it to move freely during eye movements. There is thus an allowance of upto nine millimeter proptosis before the optic nerve acts as a tether and distorts the globe. The optic nerve increases in diameter from three mm at its exit from the globe to about 4.5 mm at the orbital apex. Throughout the orbital course the nerve is surrounded by dura, arachnoid and the pia mater. The subarachnoid space is continuous with the intracranial subarachnoid space and thus contains CSF. It is supplied by recurrent branches of the short posterior ciliary arteries and capillary branches of the ophthalmic artery. Capillaries are also supplied anteriorly by branches from the central retinal artery, which pierce the optic nerve 10-15 mm from the nerve globe junction. It is also supplied by the anastomotic branch from the external carotid artery – middle meningeal artery, superficial temporal and transverse facial artery.

Intracanalicular optic nerve (intraosseous optic nerve):

The length of the intracanalicualr part is 10 mm. The optic nerve enters the optic canal through its anterior opening in the apex of the orbital roof about 5 cm posterior and 1.5 cm inferior to the supraorbital margin. This anterior opening

is called the optic foramen. The optic canal transmits the optic nerve, ophthalmic artery, some filaments of the sympathetic carotid plexus and the orbital extension of the cranial leptomeninges. This tight space in the optic nerve is particularly vulnerable to trauma[4]. Ophthalmic artery crosses the optic nerve inferiorly from medial to lateral side in the dural sheath. This nerve is supplied only by periaxial system of vessels. The pial plexus is supplied mainly from the branches from the ophthalmic artery.

Intra cranial optic nerve:

The length of the intracranial optic nerve is variable (4-15 mm) depending on the position of the chiasma in relation to the sella turcica. Its average diameter is about 4.5 to 5 mm, but it is flattened and thus is wider in the horizontal plane than in the vertical plane. Immediately above the nerve lie the anterior cerebral and anterior communicating arteries and above this lie the olfactory nerve and the frontal nerve. Just lateral to each nerve lies the internal carotid artery and ophthalmic artery arises from the internal carotid artery just below the optic nerve[5]. Just under each optic nerve and above the pituitary gland lies the planum sphenoidale. The course of the intracranial optic nerve is upward and at 45^0 angle to reach the chiasma. This part of the optic nerve is supplied by the pial plexus of vessels from branches of internal carotid artery, anterior cerebral artery or from the anterior communicating artery.

Figure 3: Optic nereve in optic canal

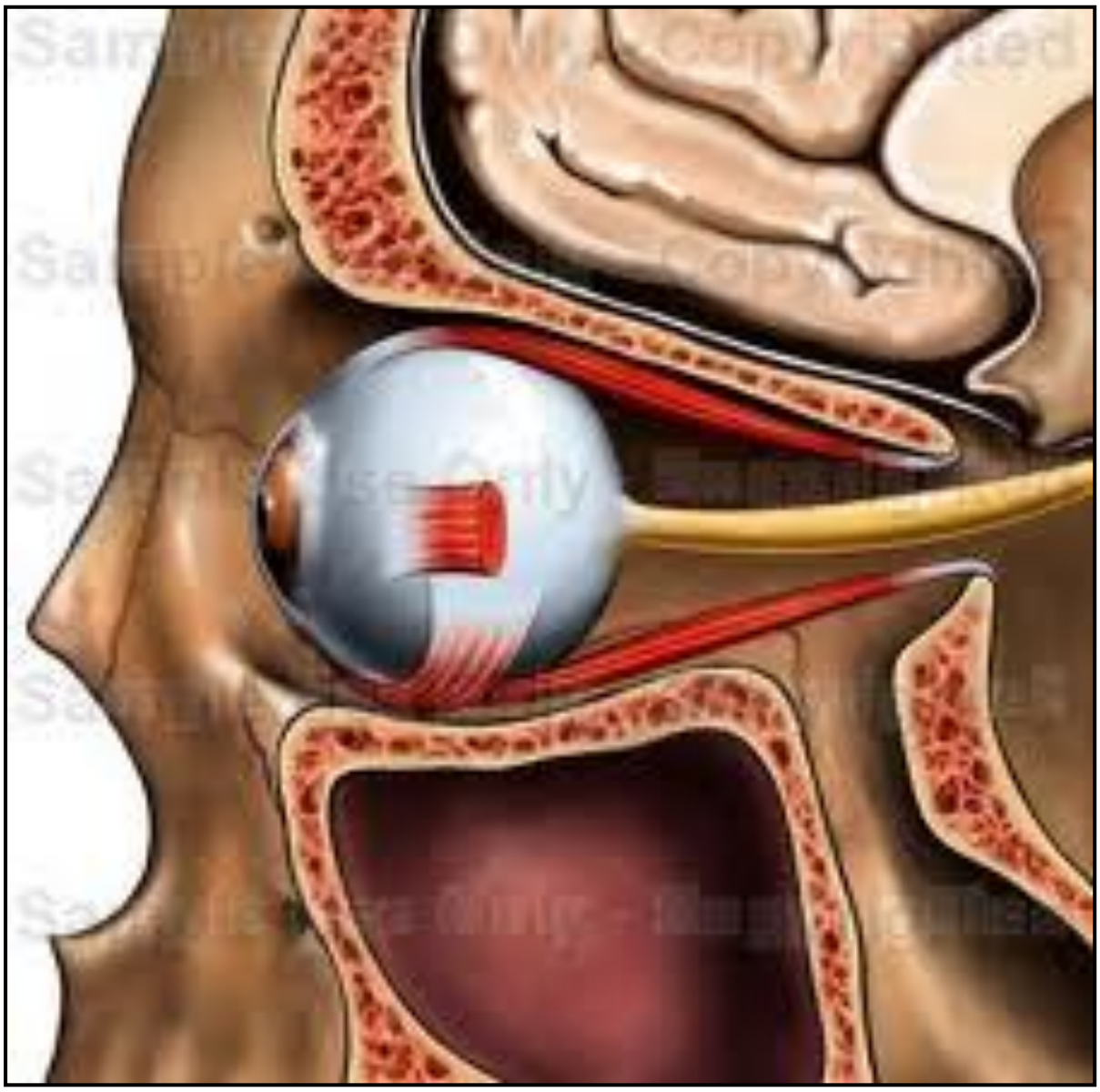


Figure 4 : Blood supply of optic nerve

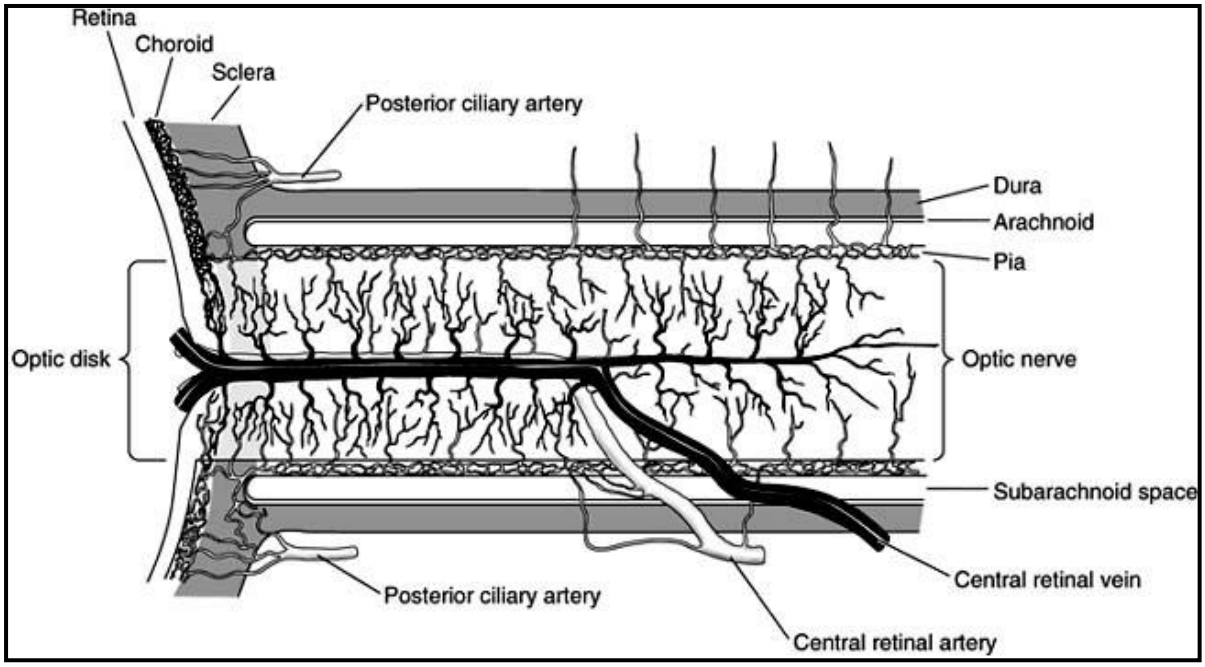
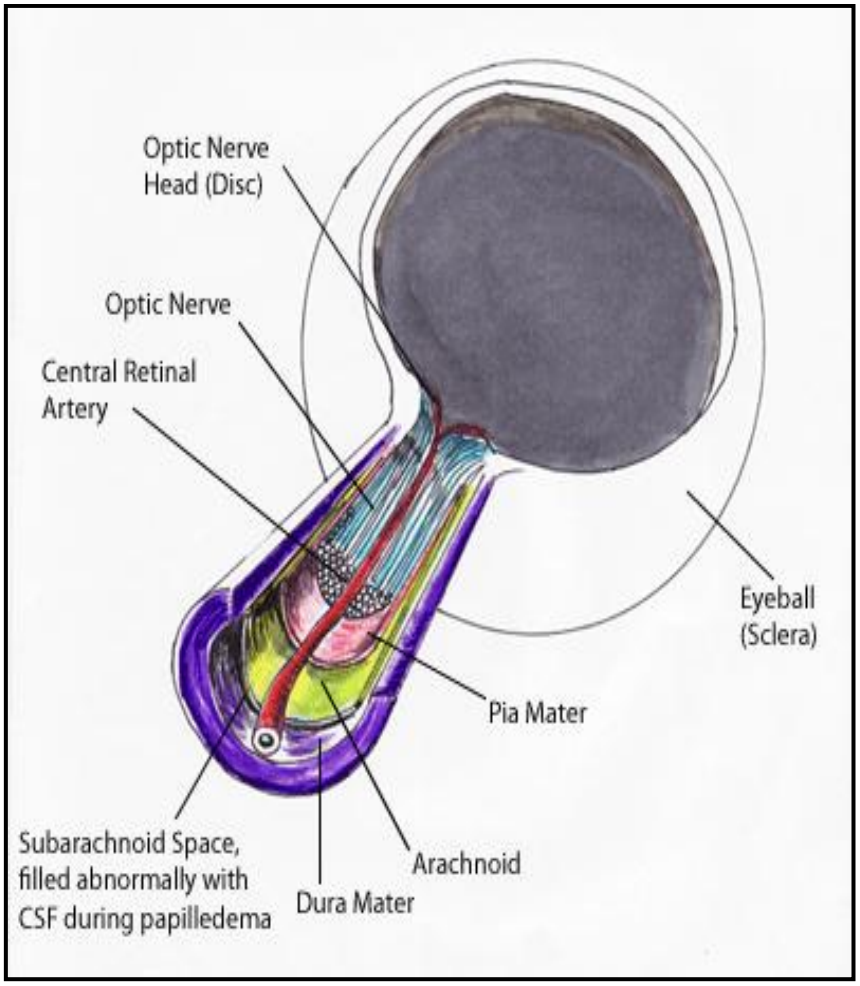


Figure 5: Covering membranes of Optic nerve



PHYSIOLOGY OF OPTIC NERVE

SYNAPTIC TRANSMISSION

Retinal ganglion cells receive synaptic input from bipolar and amacrine cells. The synapses from bipolar and amacrine cells primarily use glutamate as the major excitatory neurotransmitter. Within the retina the levels of glutamate are controlled by Müller cells, which have glutamate transporters and also contain the enzyme glutamine synthetase, which converts glutamate to the amino acid glutamine.

EXCITOTOXICITY

Retinal ganglion cells are particularly susceptible to high levels of glutamate in the extracellular space. This causes cell death mediated via overexcitation, or excitotoxicity.

AXONAL CONDUCTION

Action Potentials

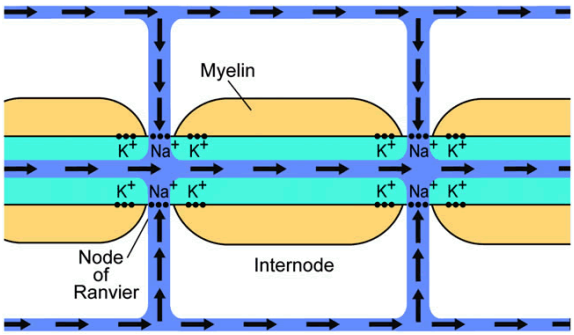
The primary function of the optic nerve is to carry information from the retina to target areas within the brain. The retinal ganglion cells and their axons are the first neurons in the transmission of visual information from the eye to use action potentials as the mechanism for transmission. Conduction down the individual axons occurs via the same biophysical mechanisms that occur in any myelinated axon.

The mechanism of axonal conduction depends on action potential. In the process of transmission of an action potential there is movement of sodium and potassium ions along their concentration gradients[6]. If action potentials continued indefinitely, the sodium and potassium concentrations would reach equilibrium across the axonal membrane, resulting in loss of their concentration gradients and blocking conduction. Therefore, to re-establish their concentration gradients, there is a relatively slow redistribution process of these ions via the Na+ K+ ATPase takes place. This is a highly energy-dependent process and will not happen if axonal metabolism is pathologically disturbed.

Role of Myelin

Myelin adds an additional complexity to axonal conduction. Myelin has two physiologic properties. It increases the resistance and decreases the capacitance of the axon. Ionic channels are not distributed uniformly along a myelinated axon. Instead, the channels are segregated into patches located within the small areas where the axon is unmyelinated. These are called nodes of Ranvier.

Conduction in a myelinated axon, therefore, becomes much faster, because the depolarization of the axon at one node leads to depolarization at the next node by the intrinsic conduction within the axon, instead of by sequential opening of channels along the axon. The depolarization, thus, “jumps” from one node to another; this is called saltatory conduction.



In cases of acquired loss of myelin, for example in idiopathic optic neuritis, a common inflammatory optic neuropathy, there is abnormal conduction because of the changes in resistive and capacitive properties of the membrane brought about by demyelination. Axonal conduction becomes slowed and in some cases may be blocked ("conduction block"), both of which result in decreased visual function. Another phenomenon peculiar to demyelination is worsening of vision with heat or exercise, called **Uhthoff's** phenomenon[7]. Increased temperature and exercise are thought to decrease the sodium channel open-time during depolarization, resulting in less charge entering the axon and a decreased likelihood that an adjacent section of demyelinated axon will be able to depolarize enough to cause opening of its own voltage-sensitive sodium channels. This leads to temperature-sensitive conduction block.

Axonal transport occurs in two directions, orthograde and retrograde. Axonal transport may be affected in disease, possibly potentiating nerve injury, such as in experimental allergic encephalomyelitis or glaucoma. Important clinical correlations of altered axonal transport are in disc edema (in which blockage of axonal transport has been observed to occur, for example, with ocular hypotony or increased intracranial pressure) and in optic neuritis.

OPTIC NEURITIS

The term optic neuritis refers to inflammatory optic neuropathy that accompanies demyelinating disease. When it is associated with swollen optic disc, it is called papillitis or anterior optic neuritis. When the optic disc appears normal, the terms retrobulbar optic neuritis or retrobulbar neuritis are used. In the absence of signs of multiple sclerosis (MS) or other systemic disease the optic neuritis is referred to as isolated, monosymptomatic, or idiopathic. The pathogenesis of isolated optic neuritis is presumed to be demyelination of the optic nerve, similar to that seen in multiple sclerosis. It is likely that most cases of isolated acute optic neuritis are a "forme fruste" of MS[7].

Optic neuritis can be caused by disorders other than MS and related demyelinating diseases. In addition, two unusual variants of optic neuritis can occur in some patients. "Neuroretinitis" is a term used to describe inflammatory involvement of both the intraocular optic nerve and the peripapillary retina. They show optic disc swelling, extensive retinal edema, haemorrhages, and hard exudates (lipid) in the macula in a star-shaped pattern. Optic perineuritis also called perioptic neuritis describes inflammatory involvement of the optic nerve sheaths without inflammation of the nerve itself associated with optic disc swelling that is unassociated with visual complaints.

Optic neuritis can be described as[8]

I) Idiopathic and primary demyelinating optic neuritis

1. Acute idiopathic demyelinating optic neuritis

2. Chronic demyelinating optic neuritis
3. Asymptomatic (subclinical) demyelinating optic neuritis
4. Neuromyelitis optica (Devic's disease)
5. Optic neuritis in myelinoclastic diffuse sclerosis

II) Secondary causes of optic neuritis

1. Viral and bacterial infections
2. After vaccination
3. Sarcoidosis
4. Syphilis
5. HIV associated optic neuritis
6. Systemic lupus erythematosus
7. Lyme disease
8. Sinus disease

Idiopathic and primary demyelinating optic neuritis

Acute idiopathic demyelinating optic neuritis

Acute demyelinating optic neuritis is far by the most common type of optic neuritis that occurs throughout the world. It is the type that is best known and understood. Much information about optic neuritis was obtained from a multicenter study called "The Optic Neuritis Treatment Trial (ONTT)".

Symptoms of optic neuritis :

The major symptoms in patients with acute optic neuritis are loss of central vision and pain in and around the affected eye.

1. Loss of central vision
2. Loss of visual field
3. Ocular or Orbital pain
4. Positive visual phenomenon

1. Loss of Central Vision:

Loss of central visual acuity is the major symptom in most cases of acute optic neuritis. Loss of vision is usually abrupt, occurring over several hours to several days. Progression for a longer period of time can occur but should make the clinician suspicious of an alternative disorder. The degree of visual loss varies widely. In some cases, visual acuity is minimally reduced, whereas in other cases, there will be complete blindness with no perception of light. The blurring of vision is predominantly central. The visual loss is monocular in most cases, but both eyes may be simultaneously affected particularly in children.

2. Loss of Visual Fields:

Not all patients with acute optic neuritis have loss of central vision. Some patients have loss of peripheral vision usually in a particular area of the visual field, such as the inferior or superior region[9].

3. Ocular or Orbital pain:

Pain in or around the eye is present in 90% of patients with acute optic neuritis. It is usually mild but it may be extremely severe than the loss of vision. It may precede or occur concurrently with visual loss. It is usually exacerbated by eye movement and generally lasts no more than few days. The pain is caused by inflammation or swelling in the optic

nerve sheaths that are innervated by small branches of the trigeminal nerve. It is hypothesized that the pain is initiated by inflammation or swelling in the optic nerve in the apex of the orbit, where the extraocular muscles are firmly attached to the sheaths of the nerve. The pain is more commonly present in retrobulbar neuritis than papillitis. The presence of pain is helpful in differentiating optic neuritis from anterior ischemic optic neuropathy, particularly when it is severe and when it occurs or worsens during movement of the eyes.

4. **Positive Visual Phenomenon:**

   Patients with optic neuritis experience positive visual phenomena, called photopsia. This may be present both at the onset of their visual symptoms and during the course of the disease. They are spontaneous flashing black squares, flashes of light, or showers of sparks[10].

Uhtoff's symptom

Following episode of optic neuritis, some patients describe transient visual blurring during a hot bath, or during emotional stress. This is most common in patients with other evidence of multiple sclerosis, but is also experienced by patients recovered from optic neuritis, and in patients with Leber's hereditary optic neuropathy. Some patients have improvement of visual symptoms with cold. Two major hypotheses regarding this phenomenon are

1. Elevation of body temperature interferes directly with axon conduction.
2. A Rise in body temperature releases a chemical substance that interferes with conduction[12].

Physical effort produced a short lasting reduction in the amplitude of the VEP and in the visual acuity in patients with multiple sclerosis with a history of Uhthoff's symptom. In patients with multiple sclerosis without a history of Uhthoff's symptom neither visual acuity nor any aspect of VEP was changed. It has been proposed that demyelinated nerves may be susceptible not only to temperature changes but also to metabolic changes in the environment.

Signs of Optic neuritis:

Visual acuity

The severity of visual acuity loss varies from a mild reduction to no light perception[13].

Colour Vision (Figure 6)

Colour vision is almost always abnormal in patients with optic neuritis and is usually more severely affected than visual acuity itself. The reduction in colour vision is much worse than would be expected from the level of acuity. So testing of colour vision may be particularly useful in diagnosing optic neuritis in patients with minor visual loss. Ishihara pseudo isochromatic colour plates could detect colour vision defects in eyes with retrobulbar optic neuritis and normal appearing optic discs. A more sensitive test for colour vision, the Farnsworth-Munsell 100-Hue test, is best for detection of optic neuropathies, including optic neuritis. The pattern of colour defects found in patients with congenital colour blindness is much different from that of acquired colour defects from optic neuropathy.

Contrast Sensitivity and Brightness sensitivity (Figure 7)

Contrast sensitivity is almost always abnormal in patients with optic neuritis. Patients with optic neuritis also have a reduced sensation in the affected eye[14]. This is tested by simply asking the patients to compare the brightness of a light shined in one eye and then another or by performing more complex testing with a flickering light the frequency of which can be varied between 50 and 0 Hz.

Visual Field

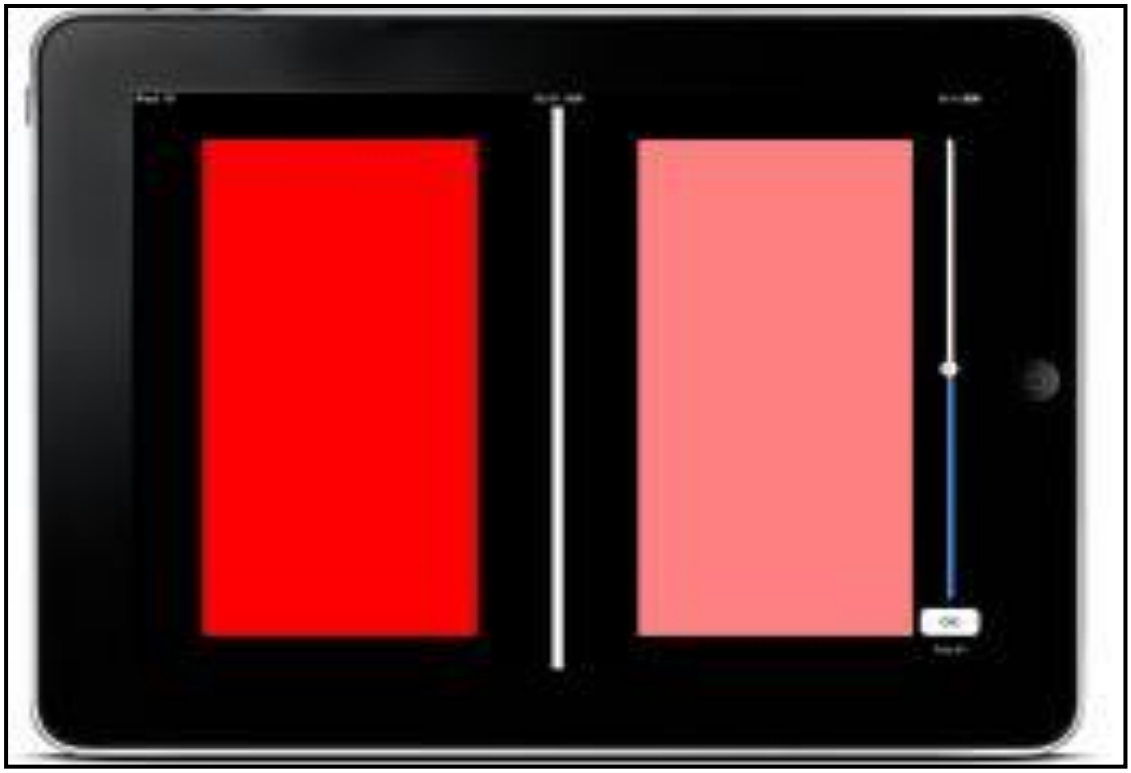
Central field is almost always affected in optic neuritis. Central and paracentral scotomas, with and without peripheral extension, comprised of 90% of field defect. Almost any type of field defect can occur in optic neuritis including an arcuate defect, a caecocentral scotoma, a superior or inferior altitudinal defect and a left or right hemianopic defect. They may also have diffuse loss of sensitivity throughout the visual field.

Pupillary Reaction

A relative afferent pupillary defect is almost always present in patients with acute optic neuritis, whether anterior or retrobulbar. The only exception to this rule is the patients with a previous attack of optic neuritis in the fellow eye or who has subclinical (asymptomatic) optic neuritis in the fellow eye[15]. Here, damage to the optic nerve in the fellow eye may offset the damage from acute optic neuritis in the affected eye (even when there is asymmetry of visual function by clinical testing), and there will be no evidence of relative afferent pupillary defect. In cases of apparent optic neuritis with no relative afferent pupillary defect by swinging flash light, this defect can be uncovered by use of neutral density filters.

Figure 6: Contrast sensitivity – reduced in optic neuritis

Figure 7: Red Desaturation



Slit lamp biomicroscopy and posterior segment inflammation:

This is almost always normal in patients with demyelinating optic neuritis. There may be few cells in the vitreous overlying the optic disc, but there is rarely a significant cellular reaction. In anterior optic neuritis (papillitis) due to causes other than demyelination such as sarcoidosis, tuberculosis, syphilis, or Lyme disease, a significant vitritis may be present.

Ophthalmoscopic appearance:

About 20 – 40% of patients with acute optic neuritis have some degree of disc swelling. In the ONTT, optic disc swelling was observed in 35% of the patient. Sometimes the disc swelling is so severe that it mimics the choked disc seen in patients with papilledema. The degree of disc swelling does not correlate with the severity of either visual acuity or visual field loss[16]. Disc or peripapillary haemorrhages are uncommon in acute optic neuritis, as opposed to AION in which they more frequently accompany disc swelling. In some patients with anterior optic neuritis, vitreous cells may be noted especially in the vitreous overlying the optic disc. When the cellular reaction is extensive etiologies other than multiple sclerosis should be considered. When macular or peripapillary hard exudates accompany the disc swelling other conditions such as neuroretinitis should be considered. Sheathing of retinal veins may occur in acute optic neuritis caused by certain conditions, including multiple sclerosis and sarcoidosis. Many of the patients with idiopathic or demyelinating acute optic neuritis have a normal optic disc in the affected eye, unless they had a previous attack of acute optic neuritis or have an ongoing chronic optic neuritis. With time, the optic disc becomes pale, even as the visual acuity and other parameters of vision improve.

The pallor may be diffuse or located to a particular portion of the disc, most often the temporal region.

Other Tests of Optic Nerve Function:

Other tests of optic nerve function including contrast sensitivity, visual evoked potential, and other psychophysical tests are almost always abnormal in patients with acute optic neuritis. Yet these tests rarely need to be performed for diagnosis of optic neuritis if other parameters are carefully evaluated. VEP is useful in distinguishing anterior optic neuritis from AION. The amplitude of the N95 peak was abnormally reduced for every eye affected with AION, whereas it remained normal in eyes with acute anterior optic neuritis[18].

Visual function in the other eye:

Several studies have reported that asymptomatic visual dysfunction may be detected in the fellow eye of a patient with acute unilateral optic neuritis. The majority of fellow eye deficits resolved over several months, suggesting that such abnormalities may be caused by subclinical acute demyelination in the fellow optic nerve.

Evaluation of patients with Acute Optic Neuritis

After through general examination and ocular examination, colour vision, field charting, and contrast sensitivity examinations are carried out. By visual acuity, pupillary reaction, colour vision, contrast sensitivity and fundus clinical diagnosis of optic neuritis is made.

Complete hemogram is done to rule out infectious and inflammatory cause of optic neuritis. Investigations like Chest X-Ray, Mantoux test are done to rule out tuberculosis. VDRL is done to rule out syphilis.

FFA are done in patients in whom diagnose is not clear to differentiate optic neuritis from AION, and other toxic neuropathies.

In VEP when there is prechiasmal demyelination, a delay in the conduction occurs which can be measured from the occipital electrodes as an abnormal P100 latency[19]. The amplitude of the P100 peak is measured; a decline in amplitude corresponds with loss of axons. While decreased amplitude implies about disease pathogenesis, activity, and recovery, it is the VEP latency that is used as a clinical test.

MRI is indicated in atypical cases, bilateral cases and recurrent cases. MRI brain shows thickened optic nerves indicating optic nerve inflammation. It shows demyelination especially in periventricular region suggestive of multiple sclerosis.

Management Recommendations for patients with presumed acute optic neuritis:

Treatment with intravenous methylprednisolone 250 mg 6^{th} hourly or 1 gm/day in a single dose for three days, followed by a two week course of oral Prednisolone 1mg/kg/day with a rapid taper for patients with acute optic neuritis if brain MRI demonstrates multiple signal abnormalities in the periventricular white matter consistent with multiple sclerosis. However oral Prednisolone in standard dosage alone should be avoided to prevent recurrence[20].

OPTIC NEURITIS TREATMENT TRIAL (ONTT)

The optic neuritis treatment trial is a multicenter controlled clinical trial that was funded by the National Eye Institute of the National Institutes of Health in the United States. In this trial 455 patients with acute unilateral optic neuritis were enrolled. The primary objective of the trial was the assessment of the efficacy of corticosteroids in the treatment of optic neuritis. It also gives some information about the clinical profile of optic neuritis, its natural history and its relationship to MS[22].

Inclusion criteria in ONTT included a clinical syndrome consistent with unilateral optic neuritis (including a relative afferent pupillary defect and a visual field defect in the affected eye), visual symptoms of eight days or less, no previous episodes of optic neuritis in the affected eye, no previous corticosteroid treatment for optic neuritis or MS, and no evidence of a systemic disease other than MS as a cause for the optic neuritis[25].

Patients were assigned to one of three treatment groups:

1. Oral prednisone (1 mg/kg/day) for 14 days
2. Intravenous methyl prednisolone sodium succinate (250 mg QID for three days) followed by oral prednisone (1mg/kg/day) for 11 days
3. Oral placebo for 14 days

Methods of examination:

Rate of visual recovery and visual outcome were assessed by

1. Snellen acuity with a retroilluminated Bailey-Lovie chart at four meters

2. Colour vision with the Farnsworth-Munsell 100-hue test[26]
3. Contrast sensitivity with the Pelli-Robson chart
4. Perimetry with the Humphrey Field Analzer (program 30-2) and Goldmann perimeter.

Complete neurological examination was performed at study entry, after six months, after one year, and then yearly. Clinically definite MS was diagnosed when a patient develops new neurological symptoms attributable to demyelination in one or more regions of the central nervous system, other than new optic neuritis in either eye, occurring atleast four weeks after the optic neuritis at study entry and lasting more than 24 hours with abnormalities documented on neurologic examination.

Observation of ONTT:

1. Most patients in all three treatment groups had a good recovery of vision.
2. After six months of follow up, the median visual acuity in each age group was 20/60 and less than 10% of the patients in each group had a visual acuity of 20/50 or worse.
3. One year after the onset of visual symptoms, there was no significant difference in mean visual cuity, color vision, contrast sensitivity, or visual field.
4. Patients treated with the regimen intravenous methyl prednisolone followed by oral prednisolone recovered vision considerably faster than patients treated with oral placebo.

5. The benefit of this treatment regimen was greatest in the first 15 days of follow up and decreased subsequently.
6. Patients treated with oral prednisone alone had an increased rate of recurrent attacks of optic neuritis in the previously affected eye and an increased rate of new attacks of optic neuritis in the fellow eye compared with patients in the other two groups. Oral Prednisolone in a dose of 1 mg/kg/day did not speed recovery of vision compared with no treatment, and did not improve ultimate visual acuity, compared with no treatment. It produced a higher rate of recurrence and new attacks of optic neuritis than no treatment[26].
7. Patients treated with intravenous followed by oral corticosteroids regimen had a reduced rate of development of clinically definite multiple sclerosis during the first two years. The clinical benefit of the intravenous treatment lessened over time such that at three years of follow up, there was no significant difference in the rate of development of MS among the treatment groups.
8. Benefit of the treatment was only seen in patients who had significantly abnormal brain MRI at the time of onset of the optic neuritis.
9. Ultimately the group concluded that there is no treatment for acute demyelinating optic neuritis that can improve the ultimate the visual prognosis compared with the natural history of the disorder[27].
10. Recurrent attacks of optic neuritis were reported within five years in 20% patients in ONTT. The likelihood of visual acuity returning to normal decreases with each recurrence.

Visual prognosis

1. Among the patients enrolled in the ONTT who received the placebo, visual acuity began to improve within three weeks of onset in 79% and within five weeks in 93%.
2. There is some correlation between the severity of visual loss and the degree of eventual recovery. Factors such as age, gender, optic disc appearance and pattern of the initial visual field defect do not appear to have any appreciable effect on the visual outcome.
3. Even those patients with improvement in visual function to normal may complain of movements induced photopsias and may have persistent visual deficits when tested using more sensitive clinical, electrophysiologic or psychophysical test.
4. The most common findings after an attack of acute optic neuritis were optic atrophy, defective colour vision and a prolonged pupil cycle time.
5. Most patients recover to normal or near normal visual acuity.
6. Persistent disturbances of colour vision are present in a high percentage of eyes with otherwise resolved optic neuritis.
7. Residual visual field defects are usually present in eyes after resolution of acute optic neuritis even when visual acuity has returned 20/20 or better. After an attack of retrobulbar optic neuritis there is normal kinetic perimetry but abnormal static perimetry with good visual acuity recovery.
8. Contrast sensitivity remains abnormal regardless of the degree of the degree of visual recovery in most eyes after resolution of acute optic neuritis. Contrast sensitivity is often abnormal in cases of multiple sclerosis in which there has not been overt acute optic neuritis. This

suggests that these eyes have subclinical optic nerve demyelination that was not necessarily detected by testing of other types of visual function.

9. The subjective visual complaints of the patients correlated better with impaired contrast sensitivity than with any other measure of visual function, including visual acuity, colour vision, and visual field.
10. Stereopsis is worse in optic neuritis compared to reduction in visual acuity in most of the cases.
11. Many patients with unilateral acute optic neuritis have a persistent relative afferent pupillary defect on the affected side, even when excellent recovery of vision has occurred.
12. Many cases of resolved optic neuritis had a persistent reduction in the amplitute of the VEP.
13. Optic disc pallor is most often present when visual recovery has been incomplete and is often present even when recovery is excellent. The pallor is usually temporal.
14. More common than optic atrophy is the development of defects in the retinal nerve fiber layer after an attack of acute optic neuritis. Most patients develop such defects, which may be diffuse, localized to the papillomacular bundle, or isolated defects in the arcuate regions.
15. Regarding subjective visual complaints, despite the often excellent measured recovery of visual function after an attack of acute optic neuritis, many patients still complain of difficulties with vision. This is mainly due to subtle abnormalities in the visual field that may be difficult to detect with standard static perimetry where patients experience abnormally rapid

disappearance of focal visual stimuli and abnormally rapid fatigue in sensitivity.

Neurologic Prognosis:

1. The risk of developing MS in patients who experience an attack of acute optic neuritis is only about 30% in patients followed 5-7 years after the attack of optic neuritis but eventually increases to about 75% in women and 34% in men with longer follow up.
2. The risk of MS was higher in women than in men.
3. The majority of patients, who develop MS after an attack of optic neuritis, do so within seven years of onset of visual symptoms.
4. Positive typing for HLA BT101-positive patients and recurrent attacks of optic neuritis were associated with an increased incidence of MS.
5. The presence of MBP in the CSF of a patient with otherwise isolated optic neuritis and no history of previous neurologic symptoms or signs is highly predictive of the development of MS.
6. Multiple oligoclonal bands in the CSF of a patient with isolated optic neuritis also is a highly predictive of future development of MS.
7. A positive family history of MS, a history of previous neurologic symptoms, and a history of a previous attack of optic neuritis in the fellow eye increased the risk of the development of multiple sclerosis.
8. The patients in whom optic neuritis is the initial manifestation of multiple sclerosis tends to have a more benign course than patients in whom multiple sclerosis presents with non visual symptoms and signs.

OPTIC NEURITIS IN MULTIPLE SCLEROSIS

Pathology

In patients with acute multiple sclerosis shows active demyelinating plaques similar to those in the brain. In such plaques, the inflammatory response is marked by perivascular cuffing of T cells and plasma cells. Initially there is swelling of nerve tissue in the area of demyelination following which the myelin sheath begins to break down into fat droplets. As the degeneration proceeds the nerve fibres are destroyed in both proximal and distal segment. After the inflammatory reaction subsides, fat laden macrophages become numerous and there is a glial proliferation. Primarily the papillomacular bundle was affected, peripheral fibres are also affected. There was an increase of cellularity of the nerve especially with respect to glial cells. The surface of optic disc showed extensive gliosis and the blood vessels on and near the disc were thickened[24]. The retina shows atrophy of the nerve fibres and ganglion cells mostly at the macula.

Pathogenesis of visual loss in optic neuritis:

Demyelination of nerve fibres that causes complete conduction block, slowing of conduction, or failure to transmit rapid train of impulses.

Relation of optic neuritis to multiple sclerosis:

Optic neuritis occurs in about 50% of the patients with MS and in about 20% of MS patients optic neuritis is the presenting feature.

Age

Younger the age at presentation with optic neuritis greater is the risk for the development of multiple sclerosis. The risk of multiple sclerosis increased with increasing age. The relative risk for development of multiple sclerosis increased by 1.7 for each decade less than 54 yrs.

Gender

The relative risk of MS is almost three times greater in women.

Features of optic neuritis[24]

Multiple sclerosis developed in only 13% of 45 patients with anterior optic neuritis compared with 60% of 30 patients with retrobulbar neuritis. The presence of severe disc swelling reduces the likelihood that MS will develop particularly when the patient has also normal MR imaging. Adult patients with bilateral simultaneous optic neuritis have the same risk of developing multiple sclerosis and patients with unilateral optic neuritis. The ultimate visual acuity after optic neuritis has no bearing on the subsequent development of multiple sclerosis.

The fellow eye have a greater risk of developing MS. Recurrent optic neuritis will increase the risk for MS.

Cerebrospinal fluid changes

Evidence of immunologic dysfunction in the CSF is common in patients with MS. Oligoclonal bands are noted in CSF.

MR imaging was found to a much stronger indicator of risk than HLA association but the combination of MR and HLA haplotype DRB1-1501 increased the predictive ability significantly.

CHRONIC DEMYELINATING OPTIC NEURITIS

Patients with chronic demyelinating optic neuritis complain of static disturbance of vision, slowly progressive loss in one or bothe eyes or occasionally stepwise loss of vision unassociated with periods of recovery. Such patients are found to have chronic optic neuritis by clinical testing like colour vision, visual field, opththalmoscopy, electrophysiological testing like VEP, psychophysical testing like contrast sensitivity or a combination of all these methods. Most patients with chronic unilateral or bilateral demyelinating optic neuritis develop visual symptoms after other signs and symptoms of MS have developed. It is the reason why the percentage of patients with MS and evidence of chronic progressive optic neuritis increases with longer follow-up[25].

ASYMPTOMATIC OR SUBCLINICAL DEMYELINATING OPTIC NEURITIS

Patients with MS have clinical or laboratory evidence of optic nerve dysfunction even though they have no visual complaints. Visually Evoked Potential seems to be particularly sensitive indicator of optic nerve and other visual sensory pathway disturbances in such patients.

NEUROMYELITIS OPTICA

Acute optic neuropathy preceded or followed within days to weeks by a transverse or ascending myelitis. It occurs commonly in children and young adults. Both sexes are equally affected. It has been reported in patients with systemic lupus erythematosis, pulmonary tuberculosis, and after chickenpox.

Both neuromyelitis optica and myelinoclastic diffuse sclerosis are variants of MS. The brain, optic nerve and spinal cord are affected by scattered lesions of demyelination that principally affect the white matter. Sometimes it may affect the grey matter also. Neuromyelitis optica cases shows evidence of a severe necrotizing myelopathy with thickening of blood vessels and absence of lymphocytic infiltration or demyelination in the spinal cord. The spinal cord is extensively affected in neuromyelitica optica. There is widespread destruction of myelin sheath and axis cylinders are also destroyed. There may be small areas of perivascular lymphocytosis in both brain and spinal cord. Formation of glial tissues occurs in mild or moderatedly severe cases. Small areas of demyelination are present in the basal ganglia in the region of red nucleus and in perivascular locations throughout the mesencephalon. Cerebellum is almost never affected in neuromyelitis optica whereas it is frequently affected in MS. Formation of cavities is common in NMO whereas it is rare in MS. Gliosis is characteristic of MS but is almost absent in NMO. Arcuate fibres located in the cerebral sub cortex are relatively unaffected in NMO.

The primary feature of NMO is visual loss either caused by damage to the anterior visual sensory pathway and paraplegia caused by damage to spinal cord.

NMO occurs as single episode without recurrences unlike MS. Patients with NMO develop mild febrile illness several days or weeks before the onset of visual or neurological manifestations. Visual loss is always bilateral. One eye is usually affected first then the other eye is affected within hours, days or rarely weeks after onset. Loss of vision is typically rapid and severe. The pupils become dilated and not reacting to light. The rapid bilateral loss of vision in NMO is in contrast to loss of vision in optic neuritis which tends to be unilateral and as severe and loss of vision in the Leber's hereditary optic neuropathy which tends to be more slowly progressive. Foci of demyelination that affect optic nerve are irregular and occur in different locations. Central scotoma is the most common field defect[24]. Most of the patients have mild swelling of the optic disc with dilatation of retinal veins with extensive peripapillary exudates.

Visual acuity usually begins to improve within one week after visual symptom. CSF analysis shows increase in concentration of protein, oligoclonal bands are rarely detected. Neuroimaging consistently shows features of demyelination of spinal cord. There is no specific treatment for NMO. Supportive care is crucial to ensure survival in patients with severe myelitis. The use of intravenous corticosteroids lessens the severity of the attack and increase the speed of recovery of both visual and motor function. Intravenous gamma globulin can also be given. The mortality rate in patients with neuromyelitis optica is as high as 50%. The visual as well as motor recovery is incomplete. Some patients are left with severe bilateral visual loss.

OPTIC NEURITIS IN MYELINOCLASTIC DIFFUSE SCLEROSIS (ENCEPHALITIS PERIAXIALIS DIFFUSA, SCHILDER'S DISEASE)

Myelinoclastic diffuse sclerosis, are non familial, do not follow an obviously viral exanthema, and are not characterized pathologically by inclusion bodies or viral particles in the CNS. It is characterised by large, sharply outlined, asymmetric focus of demyelination with severe, selective myelinoclasia that often affects an entire lobe or cerebral hemisphere. There is typically an extension across the corpus callosum and damage to the opposite hemisphere. Examination of optic nerve, brain-stem, cerebellum and spinal cord often discloses typical discrete lesions consistent with MS. The histopathological examination reveals reminiscent of MS. So it is closely related to MS.

Myelinoclastic diffuse sclerosis occurs most often in children and young adults. It is characterised by a progressive course that may be steady and unremitting or punctuated by a series of episodes of rapid worsening. Cortical blindness, irritability and central deafness can occur. The frequency of optic neuritis is less compared to MS. The fundus appearance resembles retinitis punctata albescens. Accumulation of abnormal material present in the inner aspect of internal limiting membrane. The protein content of CSF is slightly increased. IgG is often increased. Neuroimaging shows large multifocal extensive areas of demyelination. The diagnosis of myelinoclastic diffuse sclerosis is suspected when a child or young adult develops evidence of a subacute chronic progressive neurological disease with neuroimaging and laboratory evidence of focal hemispheric demyelinating disease[24].

ENCEPHALITIS PERIAXIALIS CONCENTRICA (CONCENTRIC SCLEROSIS OF BALO)

Clinically it resembles myelinoclastic diffuse sclerosis but is different pathologically. It is characterised clinically by a very rapid course during which patients develop a variety of neurological signs separated in both space and time including visual loss and diplopia. The visual loss that occurs here is usually caused by damage to post geniculate visual pathways and is characterised by homonymous field defects and cortical blindness. The pathologic changes include alternating bands of demyelination and preserved myelin in series of concentric rings in the cerebral white matter. Neuroimaging studies are initially normal. However eventually it shows multiple lesions consistent with demyelination. Treatment with systemic corticosteroids result in both immediate and long term improvement in neurological symptoms and signs.

OPTIC NEURITIS DUE TO CAUSES OTHER THAN PRIMARY DEMYELINATION

VIRAL AND BACTERIAL DISEASES:

This typically follows the onset of a viral or, less often a bacterial infection by one to three weeks. It is more common in children than in adults. It is due to immunological response producing demyelination of optic nerve. Optic neuritis may be unilateral but frequently bilateral. Parainfectious optic neuritis whether viral or bacterial may occur in patients with no evidence of neurologic manifestations or can be associated with meningitis, meningoencephalitis, or encephalomyelitis. Visual recovery following parainfectious optic neuritis is usually excellent without treatment. It is not known if corticosteroids hasten recovery in parainfectious optic neuritis. But this treatment is reasonable to consider particularly in cases with severe bilateral visual loss[25]. Optic neuritis has been reported in association with viruses like adenovirus, Coxsackie virus, CMV, hepatitis A & B, EBV, HIV type 1, measles and bacteria like anthrax, beta haemolytic streptococcus, brucellosis, cat scratch disease, pertussis and typhoid. Syphilis cause both neuroretinitis and optic perineuroretinitis. Optic neuritis in HIV patients can be retrobulbar or anterior optic neuritis.

OPTIC NEURITIS AFTER VACCINATION:

Most of the cases are bilateral. Optic neuritis has been reported after Hepatitis B, Rabies, BCG, Tetanus toxoid and Variola vaccine.

OPTIC NEURITIS IN SARCOIDOSIS:

Granulomatous inflammation of optic nerve may occur in sarcoidosis producing a typical anterior or retrobulbar neuritis.

OPTIC NEURITIS IN SYSTEMIC LUPUS ERYTHEMATOSIS:

Usually SLE is associated with typical anterior optic neuritis. The term autoimmune optic neuritis has been suggested for cases of optic neuritis in which there is serological evidence of vasculitis, progressive visual loss that tends to be responsive to treatment with systemic corticosteroids.

BILATERAL OPTIC NEURITIS

In adults bilateral optic neuritis is particularly associated with multiple sclerosis and is uncommon. Incidence of bilateral optic neuritis in MS is 10-75%. This is common in children and mostly due to viral infection.

OPTIC NEURITIS IN CHILDREN

The characteristic feature is anterior optic neuritis. It is usually bilateral and simultaneous. It occurs within one to two weeks after a known or presumed viral infection. It is less often associated with development of MS. It is mostly steroid sensitive and steroid dependent. Despite the initial poor visual acuity in optic neuritis in children, the visual prognosis appears to be quite good. Children younger than 14 years of age tended to have anterior optic neuritis whereas retrobulbar neuritis is more common in children more than 14 years of age. In younger patients, the cause of inflammation seems to be viral infection or chronic focal infection[24]. Older children often had underlying neurological disease. Optic neuritis is associated with severe disc swelling.

OBJECTIVE

This is a prospective and retrospective study done to evaluate the factors influencing the visual outcome of optic neuritis.

The objective of the study is to evaluate the effect of risk factors and the treatment given on the visual outcome of optic neuritis.

METHODOLOGY (MATERIALS AND METHODS)

Subject Selection:

All patients diagnosed as optic neuritis in RIO-GOH during May 2010 to May 2012 were included in the study.

Inclusion criteria:

All patients diagnosed as optic neuritis were studied.

Exclusion criteria:

The following patients were excluded from the study

1) Patients with Anterior Ischemic optic neuropathy
2) Patients with traumatic optic neuropathy

Methods:

In patients attending RIO GOH diagnosed as optic neuritis

1) Various risk factors associated with causation of optic neuritis were assessed.
2) Time of onset of symptoms & time when the patient presented to hospital was noted.
3) In all these patients, visual function was evaluated by Snellen's chart for visual acuity, colour vision by Ishihara pseudoisochromatic chart, assessment of contrast sensitivity Peli Robson chart and fields were tested manually by Bjerrum's screen. Pupillary assessment was done and fundus examination was done using direct ophthalmoscopy.
4) Systemic Evaluation was done for diseases causing optic neuritis.
5) The patients were started on corticosteroids as in ONTT, 3 days of I.V Methyl prednisolone at a dose of 500mg twice daily followed by Tab.Prednisolone 1mg/kg/day for 11 days and then a short taper.
6) If there were any contraindications for starting steroids, physician opinion was obtained.
7) They were also supplemented with inj.vit B1B6B12 on alternate days.
8) Related investigations were done.
9) Vision, pupil, field, colour vision, contrast sensitivity and fundus were assessed after treatment.
10) Follow up visits were given two weeks, one month and three months after the initial discharge.
11) Analysis of aetiology and visual outcome of recurrent optic neuritis was done.

Screening procedures

Procedures

1) Vision, Pupillary assessment, Fundus, Fields, Colour vision, contrast sensitivity.
2) B Scan, VEP, MRI Brain and orbit as needed

Follow up procedures/visits

Follow up visits were given two weeks, one month and three months after the initial discharge. During each follow up assessment of vision, pupil, fundus, fields, contrast sensitivity and colour vision were done.

ASSESSMENT OF PARAMETERS

Visual acuity, Pupillary assessment (Afferent pupillary defect), Fields (Central field defect) Colour vision (dyschromatopsia disproportionate to visual loss), contrast sensitivity, Fundus changes (swollen optic nerve head) and MRI (in case of multiple sclerosis).

Statistical analysis of visual outcome of optic neuritis was done in relation to the risk factors and treatment given.

OBSERVATION AND RESULTS

Totally 116 patients with acute optic neuritis were included in the study. This included first episode, recurrent episode, unilateral and bilateral cases. There were 94 unilateral patients and 22 bilateral patients were studied. Totally 138 eyes with optic neuritis were studied.

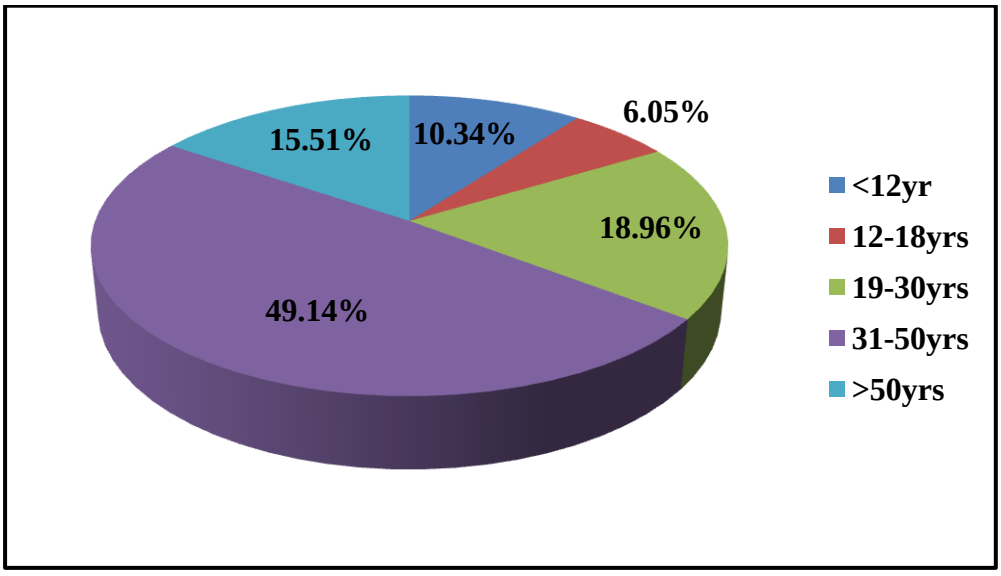
AGE DISTRIBUTION

Table 1 Age distrubution of the study patients

	No. Of patients	**% of total**
< 12 years	12	10.34%
12 -18 years	7	6.05%
19-30 years	22	18.96%
31-50 yrs	57	49.14%
>50 yrs	18	15.51%

Of the total 116 patients 12 were under the age group of <12 yrs (10.34%). Seven (6.05%) patients belong to the age group of 13 – 18 years of age. 22 (18.96%) patients were in the age group of 19-30 yrs, 57 (49.14%) in 31-50 yrs and 18 (15.51%) patients were >50 yrs of age. (Figure 1) Majority of the patients come under the age group of 31 – 50 yrs of age. Less number of patients were in the age group of 13- 18 yrs of age group. (Table 1)

Figure 1 Age distribution



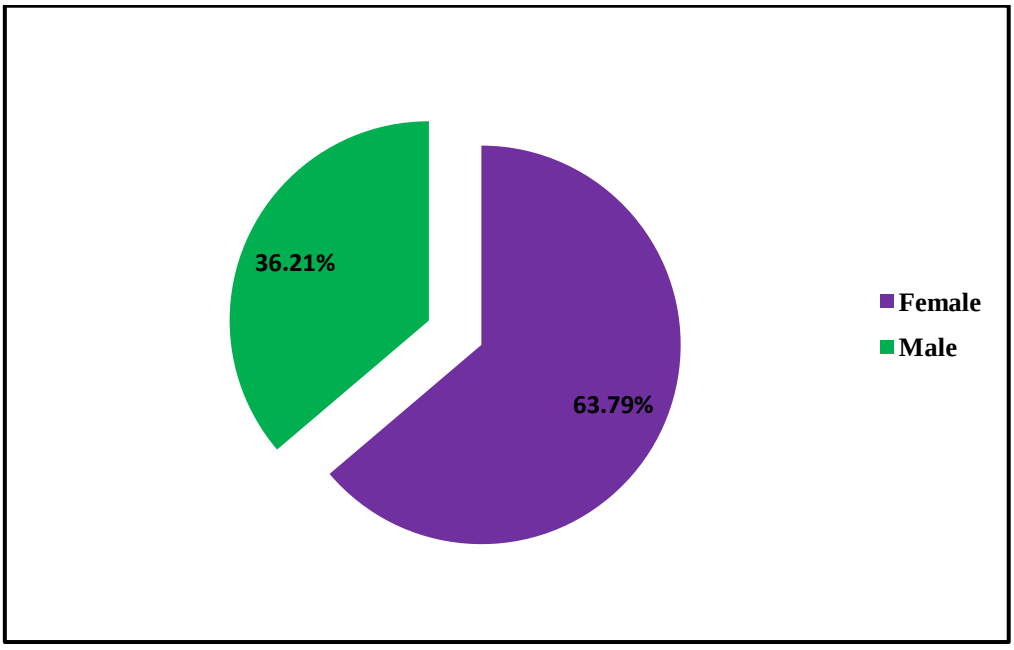
SEX DISTRIBUTION:

Table 2 Sex distribution

	No. Of patients	**% of total**
Male	42	36.21%
Female	74	63.79%
Total	116	100

Totally there were 116 patients of which 74 females (63.79%) were affected. Males were about 42 in number (36.21%). (Table 2) So females were commonly affected. (Figure 2)

Figure 2 Sex distribution



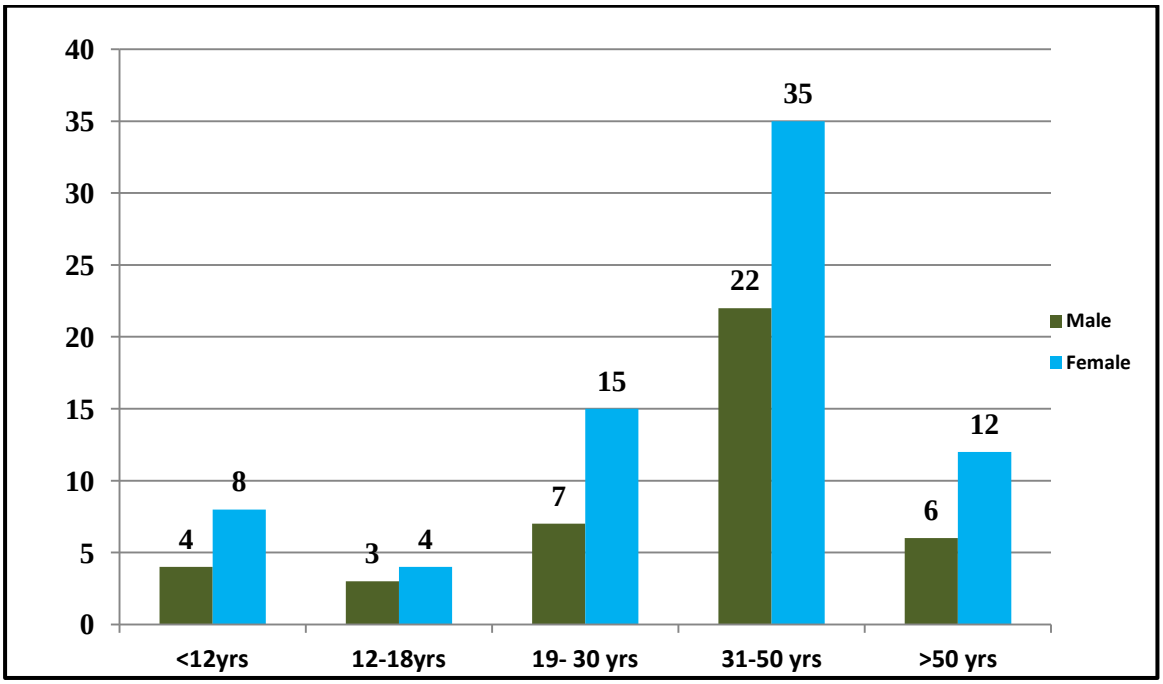
AGE SEX DISTRIBUTION

Table 3 Age Sex distribution

	Male patients	% among the age group	Female patients	% among the age group
< 12 years	4	33.33	8	66.67
12 -18 years	3	42.86	4	57.14
19-30 years	7	31.81	15	68.18
31-50 yrs	22	38.60	35	61.40
>50 yrs	6	33.33	12	66.67
	42		74	

Comparing the age and sex distribution female preponderance was seen in all age groups. (Figure3) The difference was less in 12 – 18 yrs age group. (Table 3)

Figure 3 Sex Age distribution



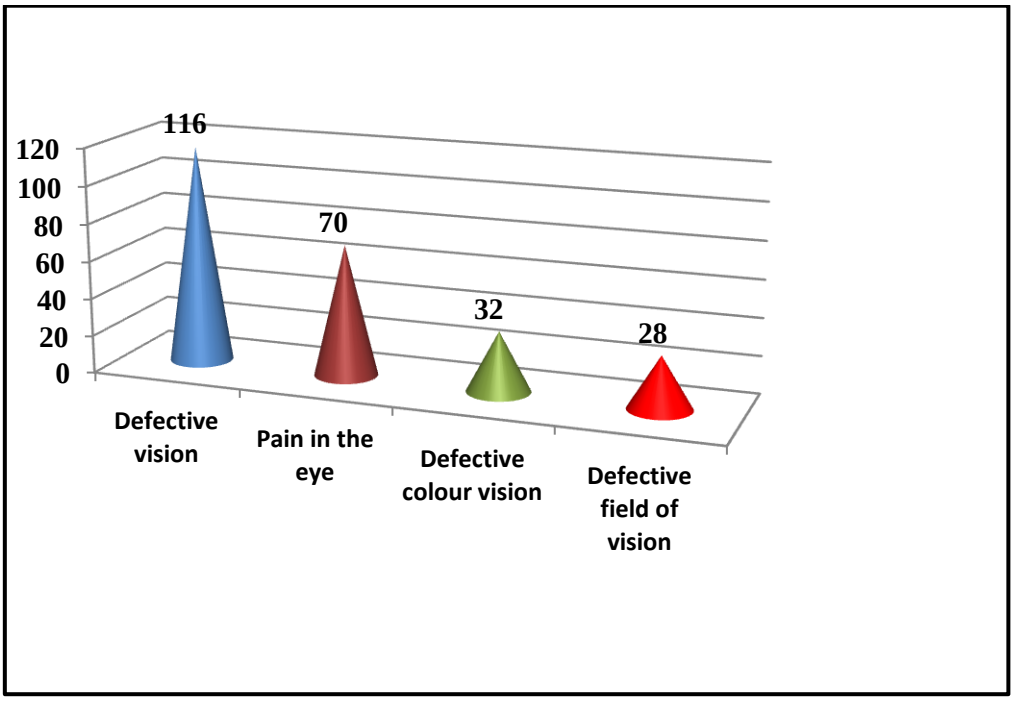
COMPLAINTS

Table 4 Complaints of the patients

	No. Of patients	% of total
Defective vision	116	100
Ocular pain	70	60.34
Defective colour vision	32	27.58
Defective field of vision	28	24.14

All the patients presented with defective vision(100%). (Figure 4) 70 patients had ocular pain (60.34%). Defective colour vision was present in 32 patients (27.58%). 28 patients had defective field of vision. (24.14%). (Table 4)

Figure 4 Complaints of the patients



RECURRENT ATTACK

Table 5 Recurrent cases of optic neuritis

	No. of cases	% of total
First episode	104	89.66
Second episode	12	10.34

Of the total 116 acute episode of optic neuritis, 12 patients had history of previous attack (10.34%). (Figure 5) Of these 12, 8 gave history of treatment taken (66.67% of recurrence) and 4 patients had not taken treatment (33.34%) (Table 5) Of the 12 recurrent cases , 7 patient had attack in the same eye itself (58.33%) and 5 had attack in the other eye (41.67%) (Figure 6).

Figure 5 Recurrent cases of optic neuritis

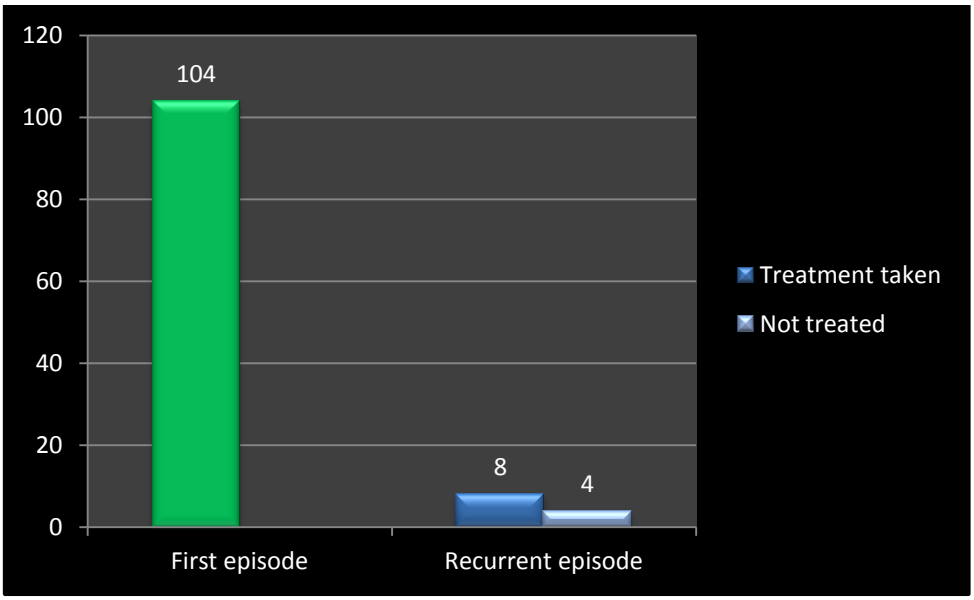
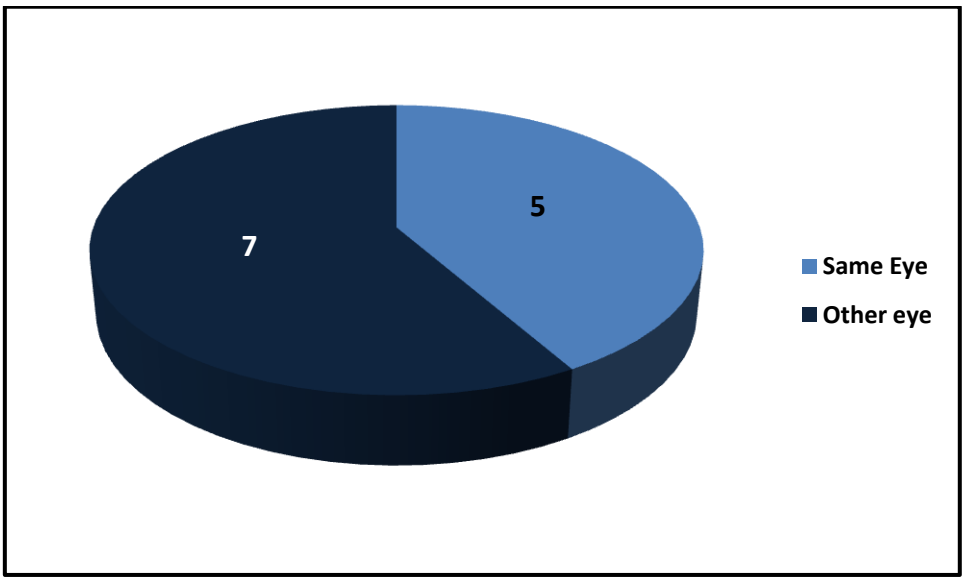


Figure 6 Pattern of Recurrence



RELEVANT PAST HISTORY:

Table 6 Relevant past history

	No. Of patients	% of total
Drug intake	1	0.86%
Diabetes mellitus	15	12.93%
Trauma	0	0
Fever	7	6.03%
vaccination	0	0
Similar episodes in family	0	0

There was history of antituberculosis treatment taken in one patient before 8 months (Table 6). There was history of fever in 7 patients (6.03%). There was no history of trauma, vaccination, family history. (Figure 7,8)

Figure 7 Relevant past history

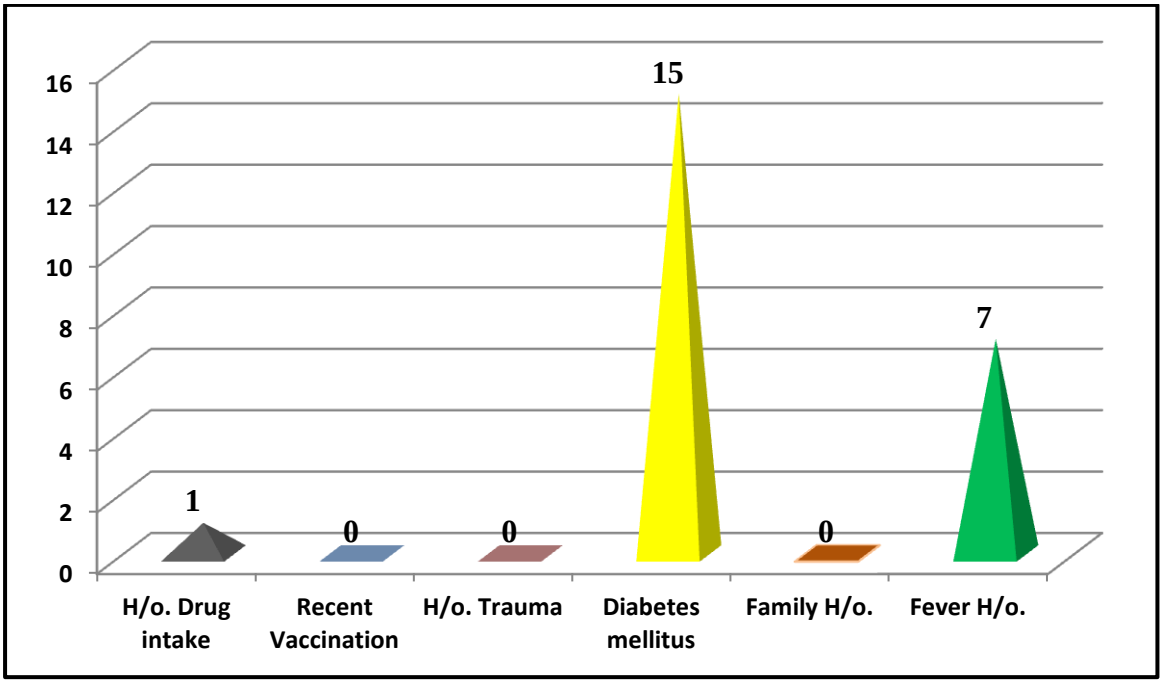
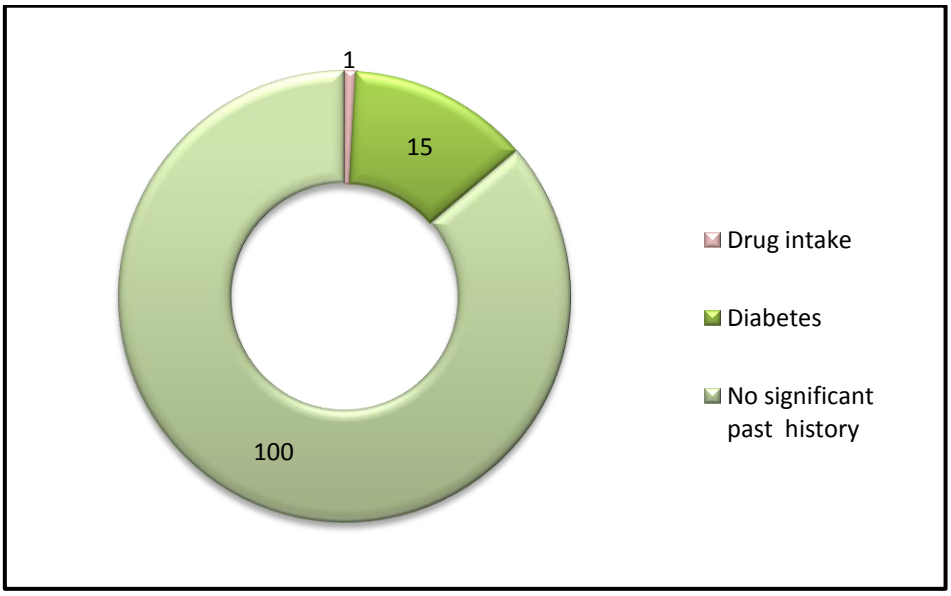


Figure 8 Past history



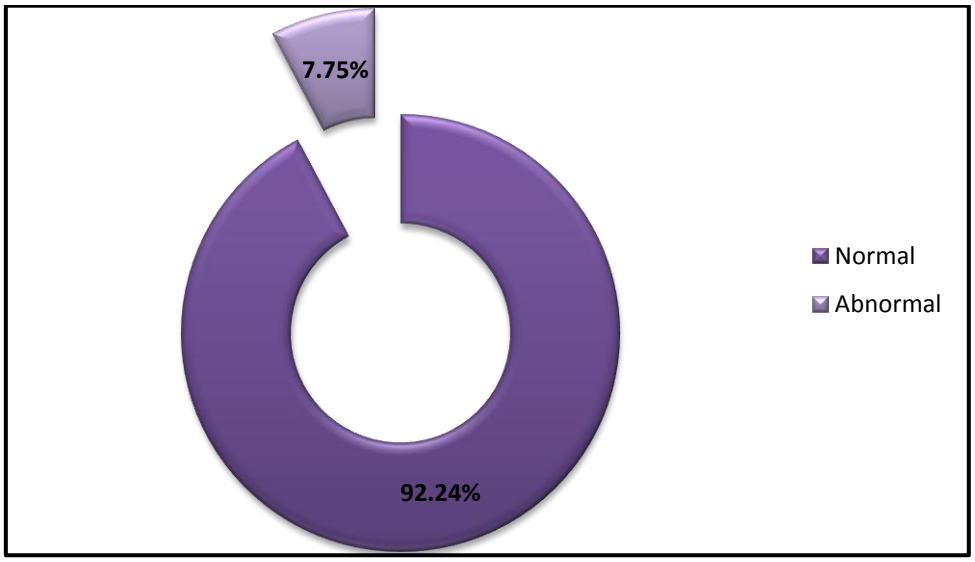
COMPLETE HEMOGRAM

Table 7 Complete hemogram

	No. of patients	% of total
Normal	107	92.24%
Abnormal	9	7.75%

Complete hemogram showed abnormal results indicative of infection in 9 patients (7.75%) and it was normal in 107 patients (92.24%) (Table 7) (Figure 9)

Figure 9 Complete hemogram

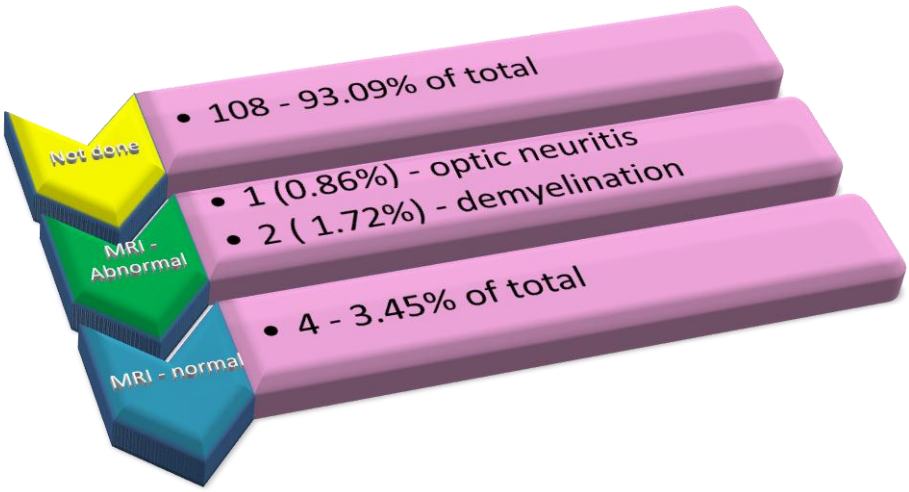


CHEST X RAY

Chest X-ray was normal in all the patients.

MRI BRAIN

Figure 10 MRI of the study population



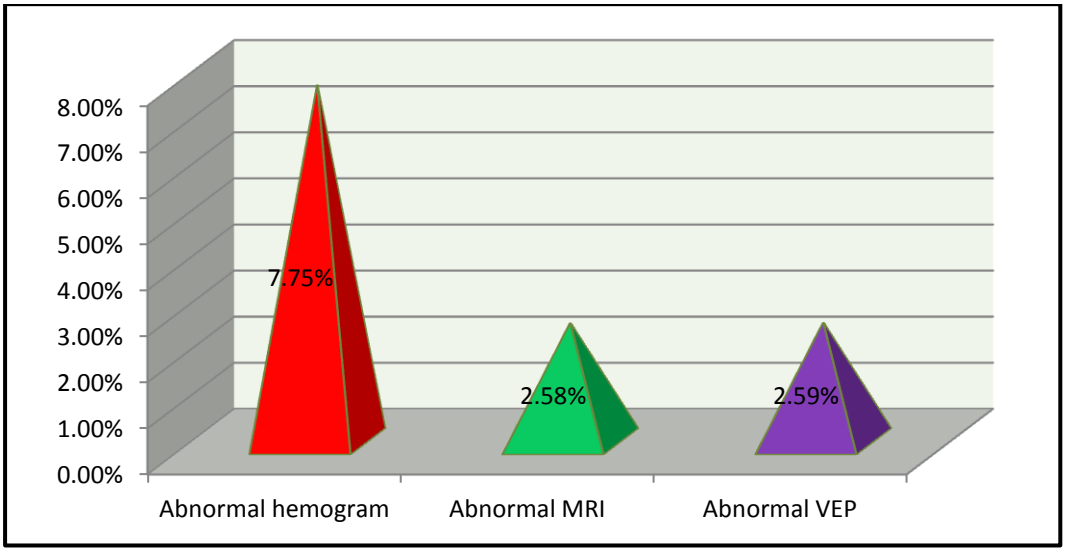
MRI brain was advised in atypical cases such as bilateral optic neuritis, recurrence optic neuritis and who do not respond to treatment. But due to cost factor, MRI was done in seven patients. Of them, 4 were found to be normal (3.45%), in 2 cases there was suggestion of demyelination (1.72%) and in one patient there was finding of optic neuritis (0.86%). (Figure 10)

VEP

VEP was done in three patients to confirm the diagnosis of optic neuritis. All the three cases showed features of demyelinating optic neuritis.

ABNORMAL INVESTIGATIONS:

Figure 11 Abnormal investigations



Abnormal hemogram was noted in 7 patients (7.75%), MRI was abnormal in 3 patients (2.58%) and VEP was abnormal in 3 patients (2.58%). (Figure 11)

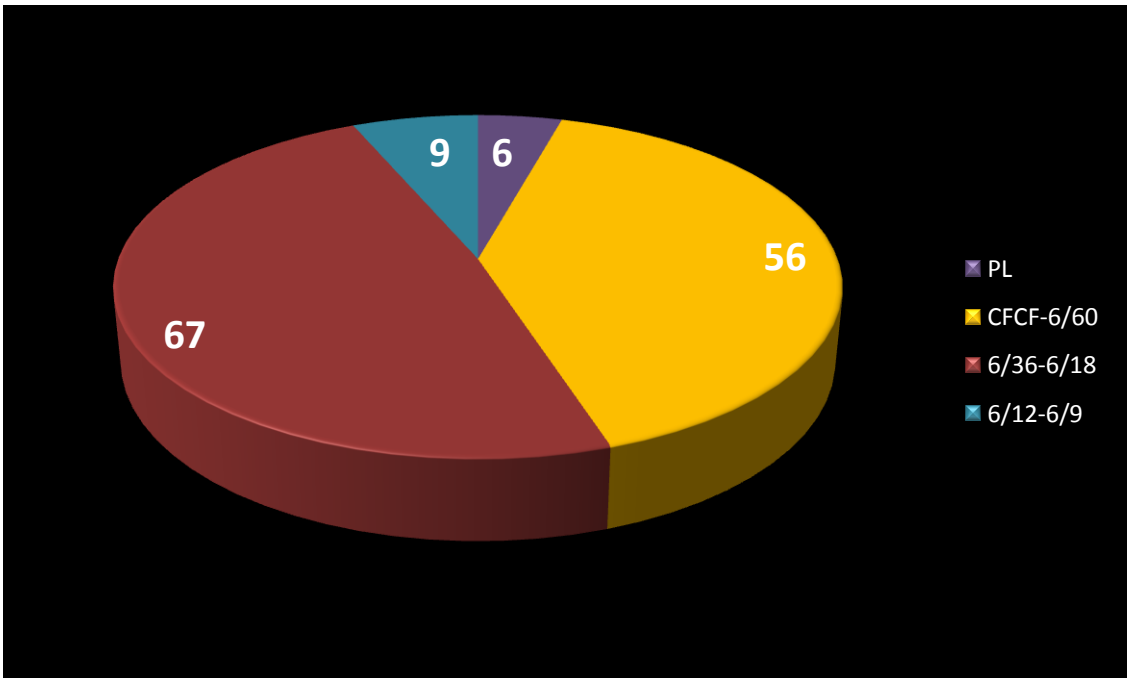
VISUAL ACUITY AT THE TIME OF PRESENTATION

Table 8 Visual acuity at the time of presentation

	No. Of cases	% of total eyes
PL+	6	4.35
CFCF – 6/60	56	40.57
6/36 – 6/18	67	48.56
6/12 – 6/9	9	6.52
	138	100

At the time of presentation 6 had only perception of light (4.34%), 56 had vision between CFCF and 6/60 (40.47%), 67 had vision of 6/36-6/18 (48.55) and 9 had vision of 6/12 – 6/9 (6.52%). (Table 8) Majority of the patients were with vision of 6/36-6/18. Only very few patients had vision of PL and 6/12-6/6. (Figure 12)

Figure 12 Visual acuity at the time of presentation



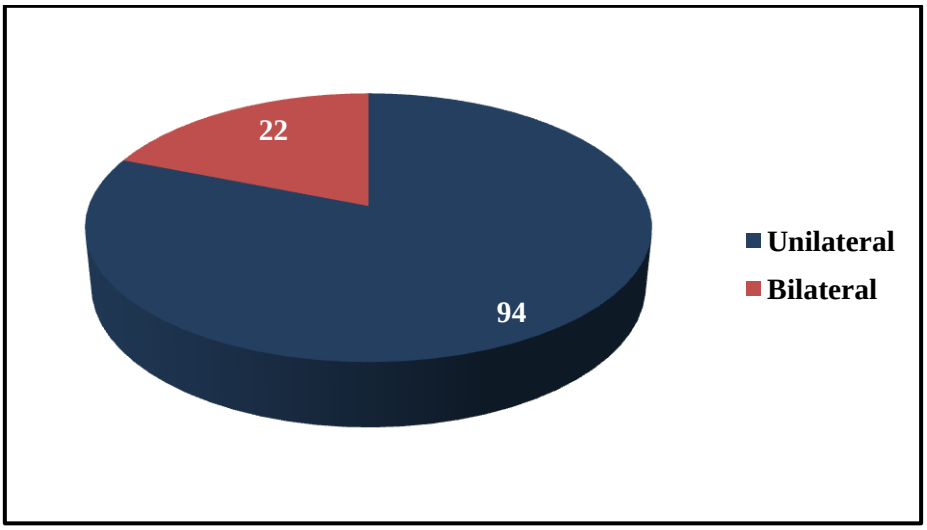
LATERALITY

Table 9 Laterality of the eyes

	No. of patients	% of total
Unilateral	94	81.03
Bilateral	22	18.97

94 patients presented with unilateral optic neuritis (81.03%) and 22 presented with bilateral optic neuritis (18.97%). (Table 9) (Figure 13)

Figure 13 Laterality of the eyes

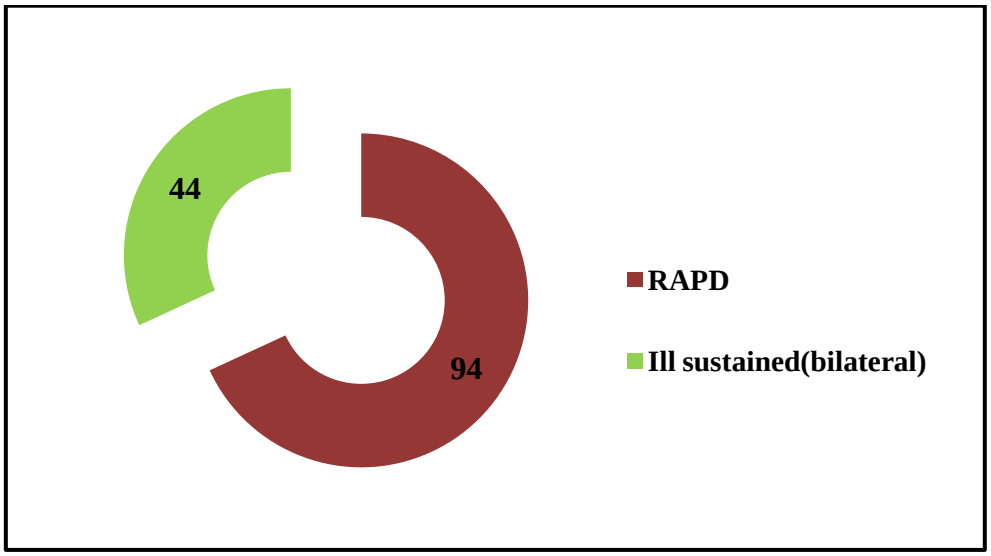


PUPILLARY REACTION AT THE TIME OF PRESENTATION

Table 10 Pupillary reaction at the time of presentation

	No. Of cases	% of total
Normal	0	0
RAPD	94	68.12
Ill sustained	44	31.88
	138	**100**

Figure 14 Pupillary reaction at the time of presentation



Among the 116 patients, RAPD was noted in 94 persons (81.03%) and ill sustained reaction was noted in bilateral cases (18.93%) (Figure 14). Pupillary abnormality was noted in all the study patients (Table 10).

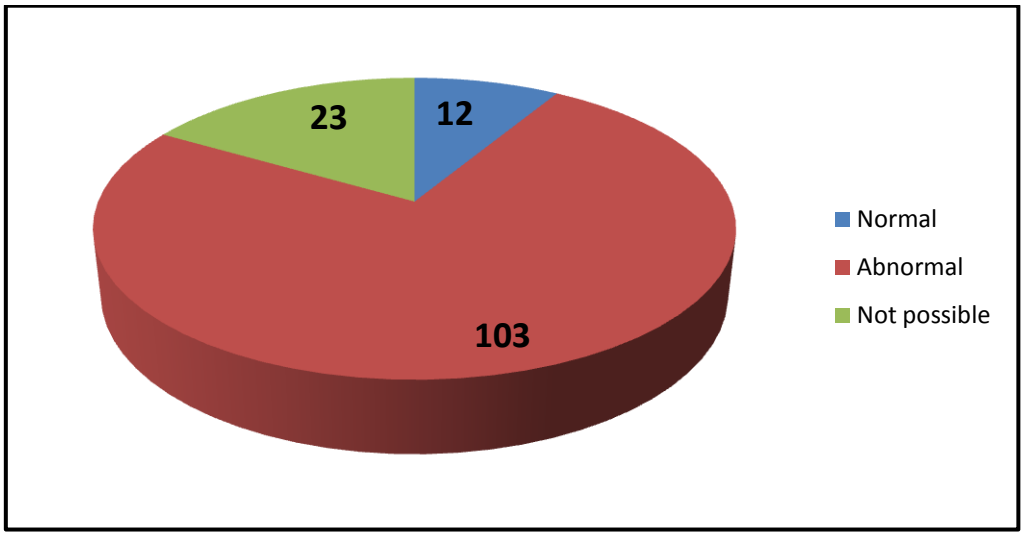
COLOUR VISION AT PRESENTATION

Table 11 Colour vision at presentation

	No. Of cases	% of total
Normal	12	8.69
Defective	103	74.74
Not possible	23	16.67
	138	100

Colour vision abnormality (red desaturation) was noted in 103 patients (74.74%). Colour vision examination was not possible in 23 patients (16.67%) due to poor vision. It was normal in 12 patients (8.69%) (Figure 15). Most of the cases showed abnormal colour vision at the time of presentation (Table 11).

Figure 15 Colour vision at presentation



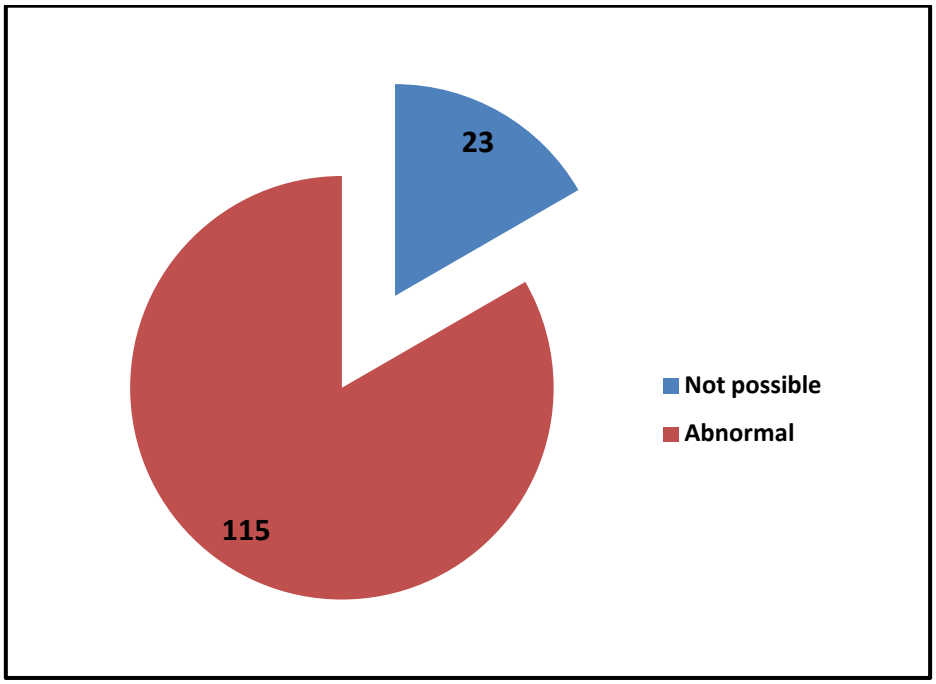
CONTRAST SENSITIVITY

Table 12 Contrast sensitivity

	No. Of cases	**% of total**
Normal	0	0
Defective	115	83.33
Not possible	23	16.67
	138	100

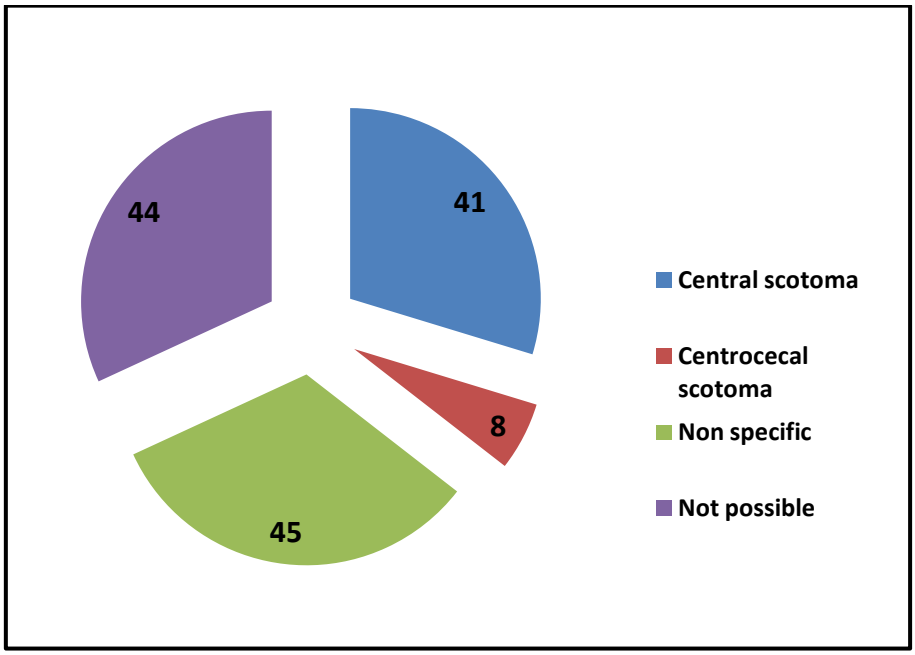
Contrast sensitivity could not be tested in 23 patients due to poor vision (16.67%). (Figure 16) In all possible patients contrast sensitivity was abnormal (83.33%) (Table 12)

Figure 16 Contrast sensitivity



FIELDS AT THE TIME OF PRESENTATION

Figure 17 Fields at time of presentation



At the time of presentation, field charting was not possible in 44 patients (31.88). Central scotoma was present in 41 patients (29.71%) . Centrocaecal scotoma and non specific changes were noted in 8 (5.80%) and 45 (32.61%) respectively (Table 13). Central scotoma and non specific changes were more common (Figure 17).

Table 13 Fields at time of presentation

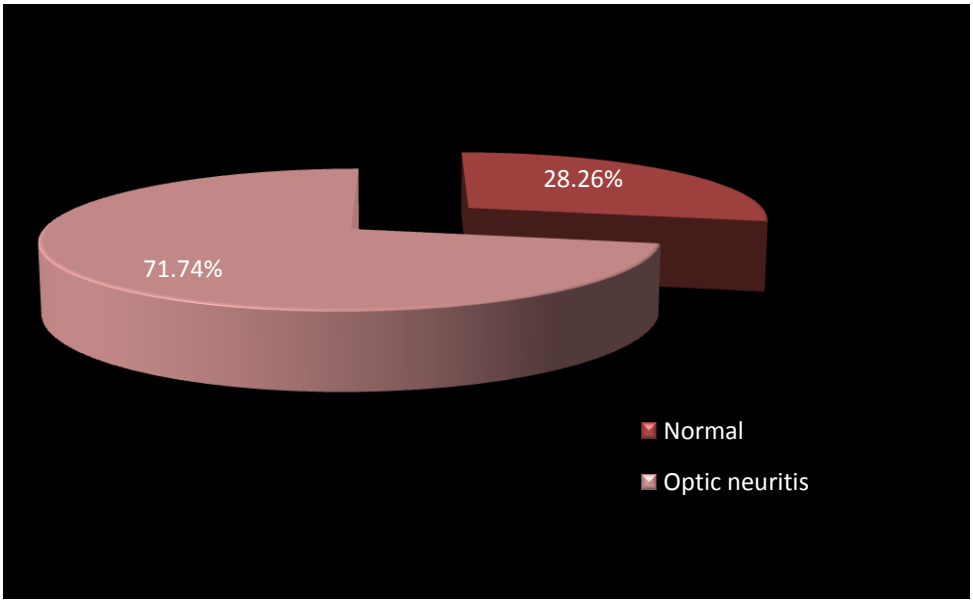
	No. Of cases	**% of total**
Normal	0	0
Central scotoma	41	29.71
Centrocecal scotoma	8	5.80
Non specific	45	32.61
Not possible	44	31.88

FUNDUS AT THE TIME OF PRESENTATION

Table 14 Fundus at time of presentation

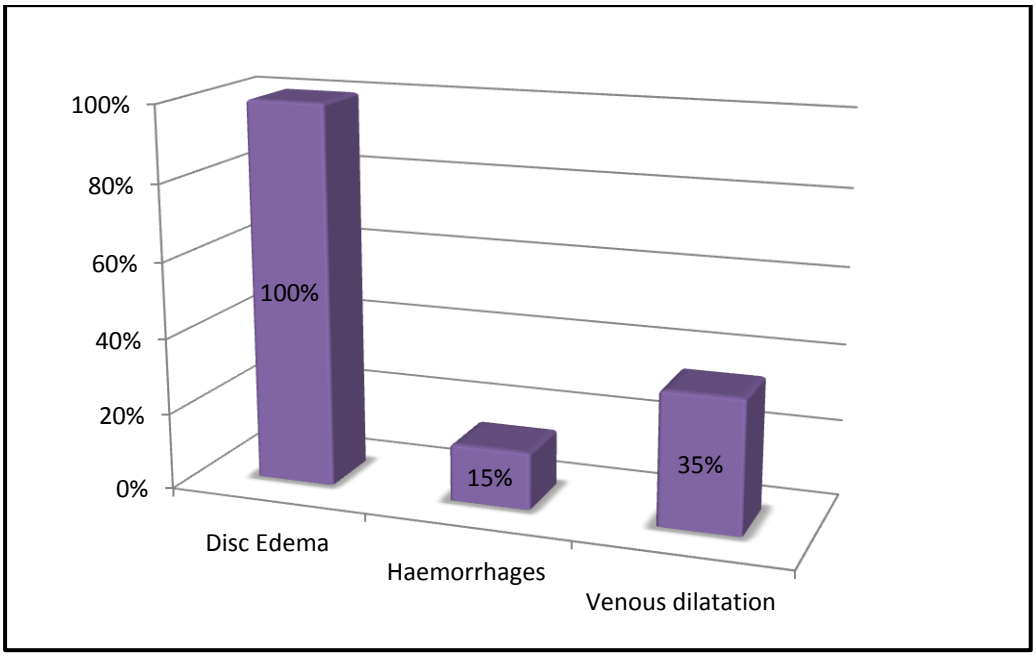
	No. Of cases	**% of total**
Normal	39	28.26
Abnormal	99	71.74
Temporal pallor	0	0
	138	100

Figure 18 Fundus at time of presentation



Fundus showed features of anterior optic neuritis including optic disc edema, haemorrhages and venous dilatation in 99 patients (71.74%). Fundus was normal in 39 patients (28.26%). Anterior optic neuritis was more common than retrobulbar neuritis (Table 14) (Figure 18).

Figure 19 Optic neuritis fundus



In abnormal fundus, disc edema was present in all cases. Haemorrhages was noted in 15% and venous dilatation in 35% (Figure 19).

ONTT:

All patients were given ONTT.

POST TREATMENT VISUAL ACUITY – ONE WEEK

Table 15 Post treatment visual acuiy – one week

Post treatment Visual Acuity one week		
VISUAL ACUITY	**No. Of patients**	**Percentage of patients (%)**
PL+	0	0
CFCF – 6/60	0	0
6/36 – 6/18	32	23.18
6/12 – 6/9	42	30.44
6/6	64	46.38

After one week of treatment 32 patients had visual acuity of 6/36 – 6/18 (32%), 42 were with visual acuity of 6/12 – 6/6 (30.44%) and 64 (46.38%) had visual acuity of 6/6. (Table 15)

POST TREATMENT COLOUR VISION – ONE WEEK

Table 16 Post treatment Colour vision – one week

	No. Of cases	**% of total**
Normal	36	26.09
Defective	102	73.91
Not possible	0	0
	138	100

Post treatment, colour vision became normal in 26% patients and remained defective in 73% of patients. (Table 16)

POST TREATMENT PUPIL – ONE WEEK

Table 17 Post treatment pupil – one week

	No. Of cases	**% of total**
Normal	84	60.87
RAPD	0	0
Ill sustained	54	39.13
	138	100

After one week of treatment, pupillary reaction was ill sustained in 54 eyes (39.13%). It was normal in 84 eyes (60.87%). (Table 1)

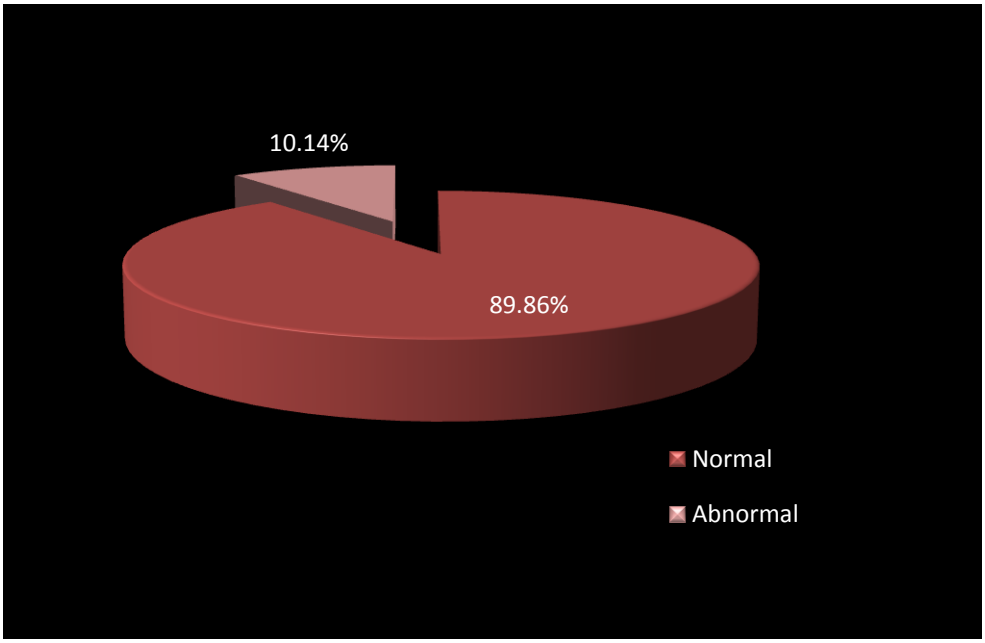
POST TREATMENT FUNDUS – ONE WEEK

Table 18 Post treatment fundus – one week

	No. Of cases	**% of total**
Normal	124	89.86
Abnormal	14	10.14
Temporal pallor	0	0
	138	100

One week after treatment fundus was normal in 89% of patients. It remained abnormal in 10% of patients (Table 18) (Figure 20).

Figure 20 Post treatment fundus – one week



POST TREATMENT – ONE MONTH

One month after presentation 85 patients (61.59%) had visual acuity of 6/6 (Table 19). Only 12 patients had visual acuity less than 6/18. After one month of treatment, Colour vision remained defective in 63% of individuals (Table 20). Pupillary abnormality persisted in 26% of patients (Table 21). Fundus abnormality persisted in only 2 patients (1.45%) (Table 22) (Figure 21).

Table 19 Post treatment visual acuity – one month

Post Operative Visual Acuity – one month		
VISUAL ACUITY	**No. Of patients**	**Percentage of patients (%)**
6/36 – 6/18	12	12.69
6/12 – 6/9	41	29.72
6/6	85	61.59
	138	100

POST TREATMENT COLOUR VISION – ONE MONTH

Table 20 Post treatment colour vision – one month

	No. Of cases	% of total
Normal	50	36.23
Defective	88	63.77
Not possible	0	0
	138	100

POST TREATMENT PUPIL – ONE MONTH

Table 21 Post treatment pupil – one month

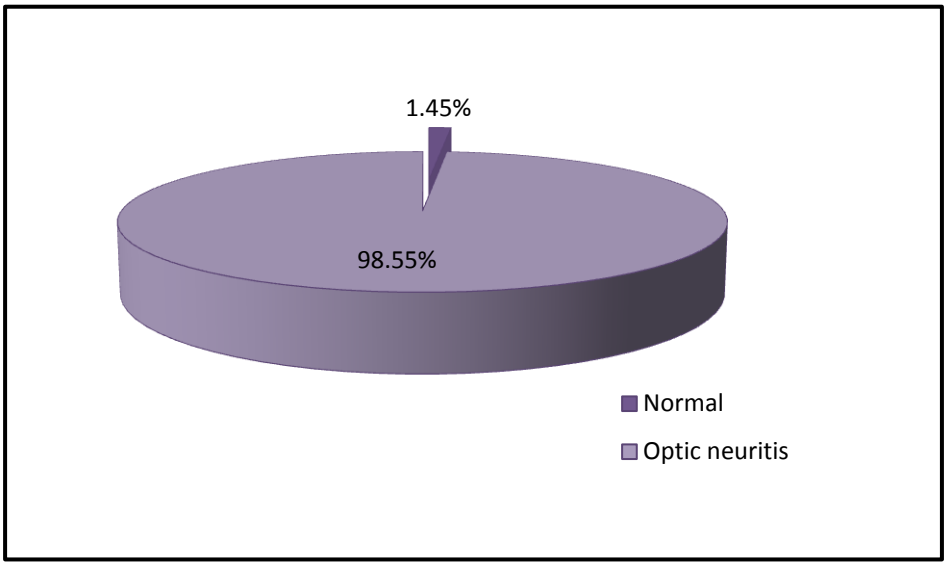
	No. Of cases	% of total
Normal	101	73.19
RAPD	0	0
Ill sustained	37	26.81
	138	100

POST TREATMENT FUNDUS – ONE MONTH

Table 22 Post operative fundus – one month

	No. Of cases	% of total
Normal	136	98.55
Abnormal	2	1.45
Temporal pallor	0	0
	138	100

Figure 21 Post treatment visual acuity – one month



POST TREATMENT VISUAL ACUITY – THREE MONTHS

After three months of treatment, 65% had vision of 6/6 (Table 23). But colour vision defect persisted in 60% of the patients (Table 24). Pupillary abnormality persisted in 23% of patients (Table 25). Fundus showed temporal pallor in 11 patients (7.97%) (Table 26) (Figure 22)

Table 23 Post treatment visual acuity – three months

Post treatment Visual Acuity – three months		
6/36 – 6/18	8	5.79
6/12 – 6/9	40	28.98
6/6	90	65.23

POST TREATMENT COLOUR VISION – THREE MONTHS

Table 24 Postoperative Colour vision – three months

	No. Of cases	**% of total**
Normal	55	39.86
Defective	83	60.14
Not possible	0	100
	138	**100**

POST TREATMENT PUPIL – THREE MONTHS

Table 25 Postoperative pupil – three months

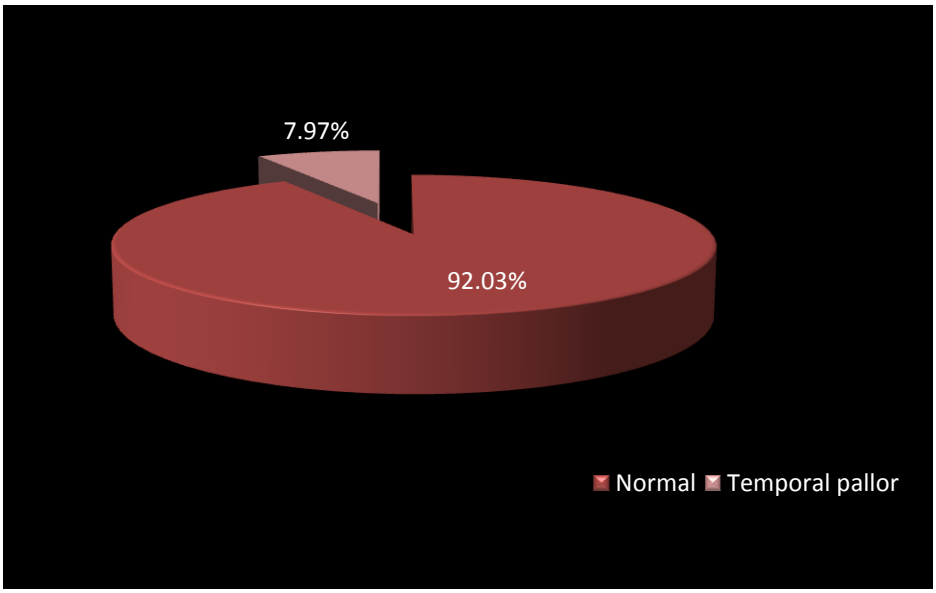
	No. Of cases	**% of total**
Normal	106	76.81
RAPD	0	0
Ill sustained	32	23.19
	138	100

POST TREATMENT FUNDUS – THREE MONTHS

Table 26 Postoperative Fundus – three months

	No. Of cases	**% of total**
Normal	127	92.03
Abnormal	0	0
Temporal pallor	11	7.97
	138	**100**

Figure 22 Post treatment Fundus – three months



COMPARISION OF PARAMETERS ONE WEEK, ONE MONTH, THREE MONTHS POST TREATMENT WITH PRE-TREATMENT PARAMETERS

Visual acuity improvement was significant in one and three months of treatment. Colour vision defect persisted in significant percent of patients even after three months. Pupillary reaction remained abnormal in 23% of patients. Fundus remained abnormal only in 1% after one month. Fundus showed temporal pallor in 11 patients after three months (Figure 23).

Table 27 Visual acuity comparison before and after treatment

		Post treatment		
	Presentation	**One week**	**One month**	**Three months**
PL+	4.35	-	-	-
CFCF – 6/60	40.57	-	-	-
6/36 – 6/18	48.56	23.18	12.69	5.79
6/12 – 6/9	6.52	30.44	29.72	28.98
6/6	-	46.38	61.59	65.23
P value *		0.030**	0.006***	0.002***

*by chi square test

**significant at $p < 0.05$

***significant at $p<0.01$

Colour vision comparison before and after treatment

Table 28 Colour vision comparison before and after treatment

		Post treatment		
	Presentation	**One week**	**One month**	**Three months**
Normal	8.69	26.09	36.23	39.86
Defective	74.74	73.91	63.77	60.14
Not possible	16.67	0	0	0
P value *		0.232	0.112	0.105

*by chi square test

Pupillary reaction compared prior and after treatment

Table 29 Pupillary reaction comparison before and after treatment

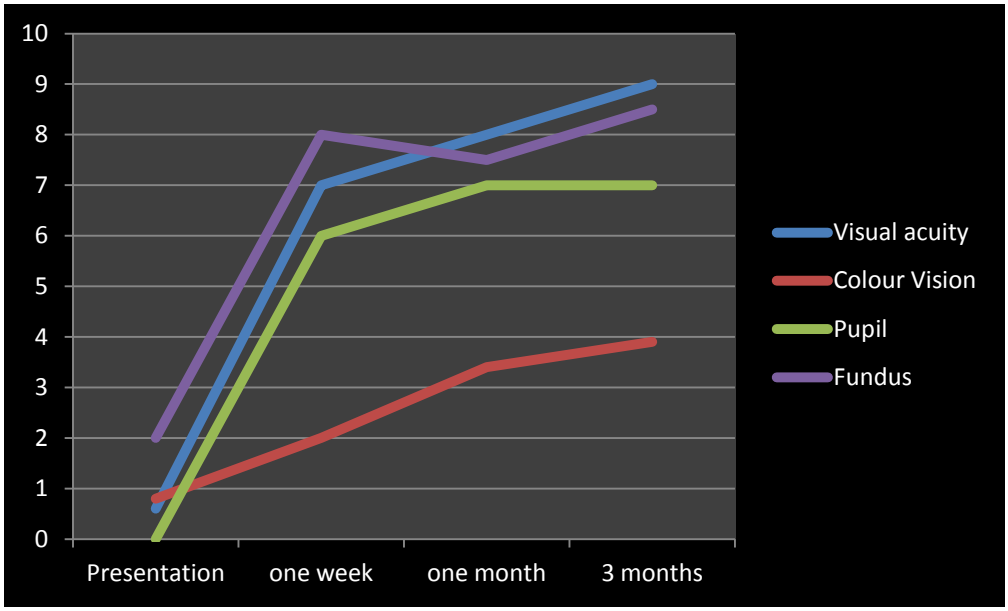
		Post treatment		
	Presentation	**One week**	**One month**	**Three months**
Normal	0	60.87	73.19	76.81
RAPD	68.12	0	0	0
Ill sustained	31.88	39.13	26.81	23.19

Fundus changes compared prior and after treatment

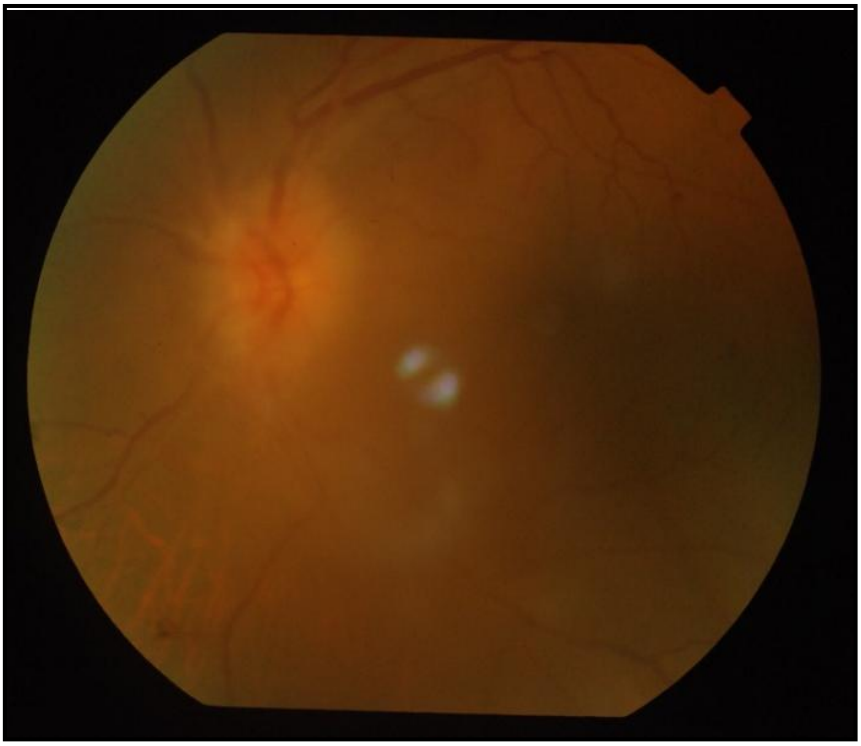
Table 30 Fundus before and after treatment

		Post treatment		
	Presentation	**One week**	**One month**	**Three months**
Normal	28.26	89.86	98.55	92.03
Abnormal	71.74	10.14	1.45	0
Temporal pallor	0	0	0	7.97

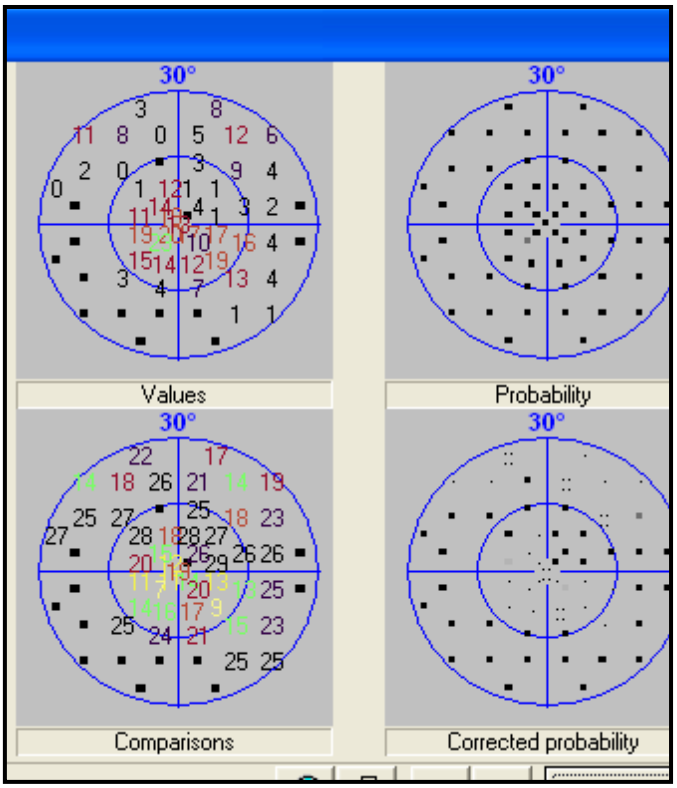
Figure 23 Visual parameters before and after ONTT



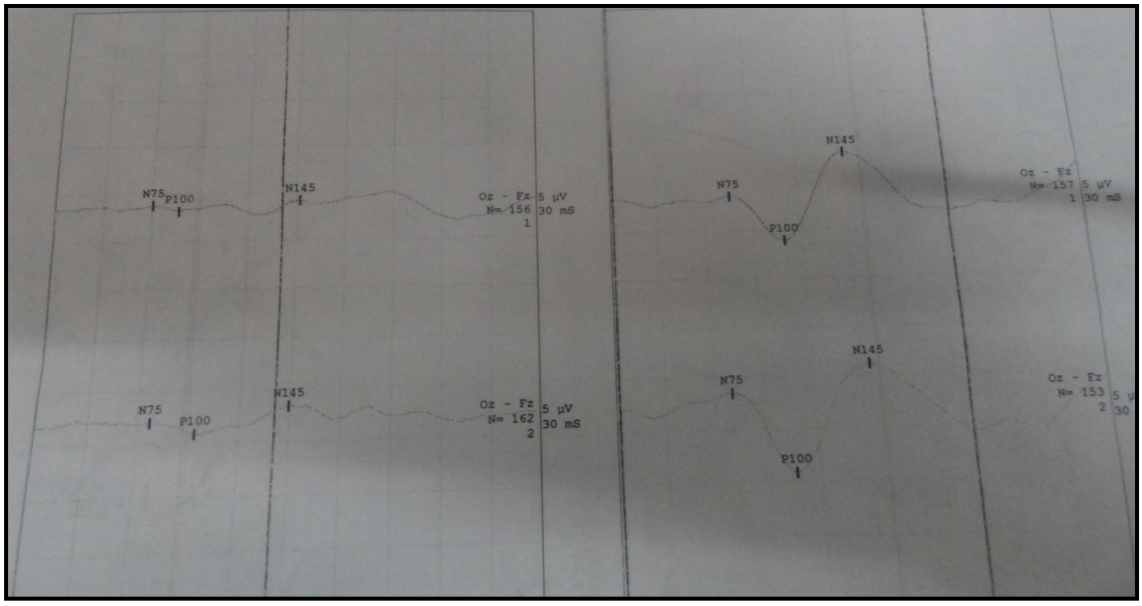
Fundus picture of patient no 106 showing optic disc oedema



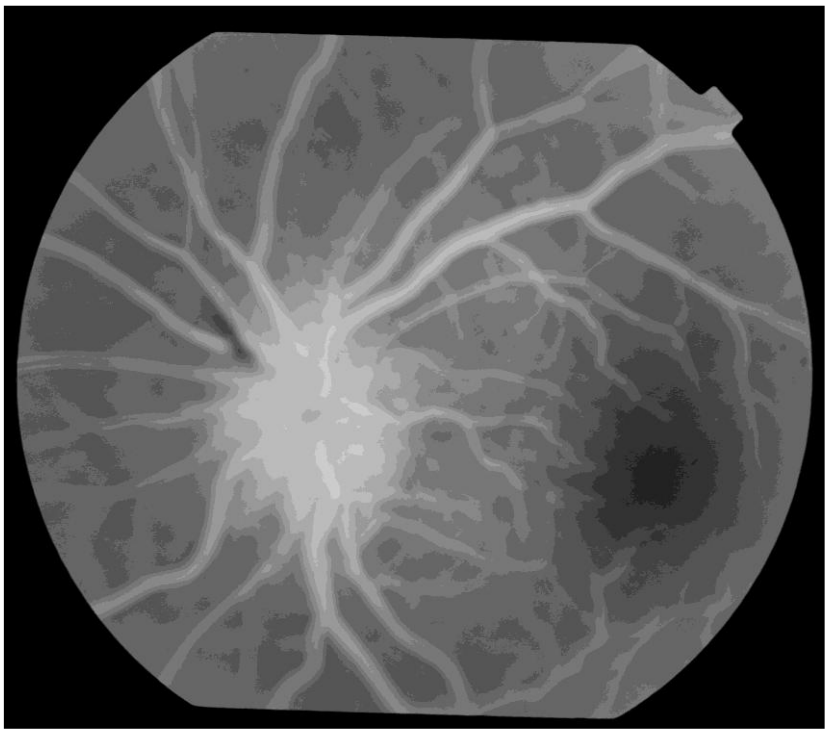
AP of patient 106 showing non specific defect



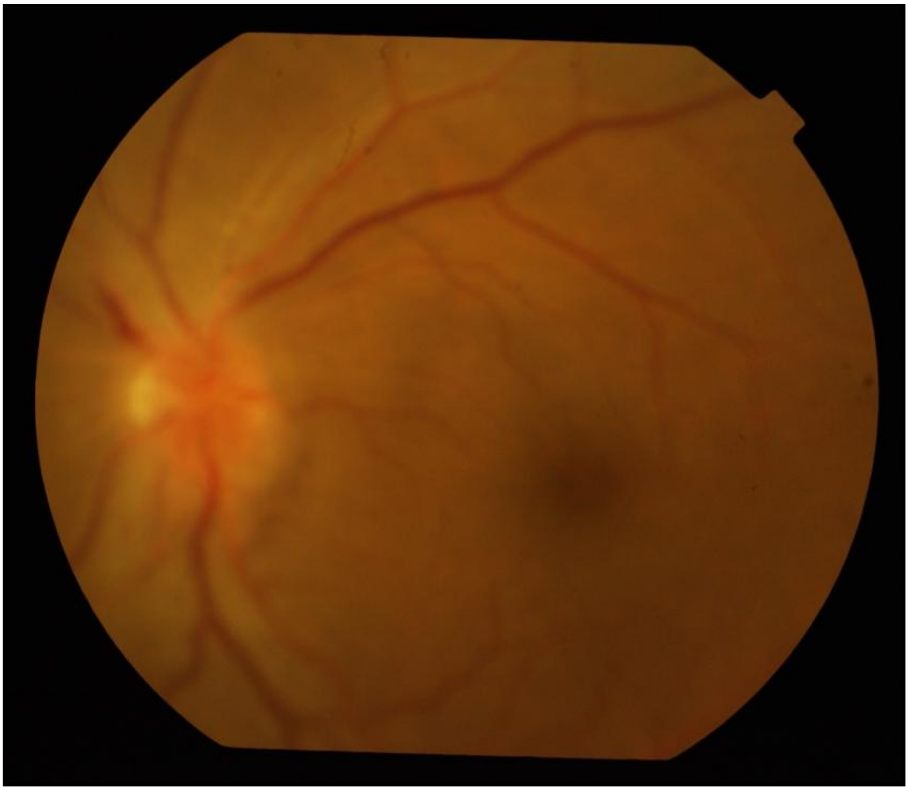
VEP of patient No.106 showing reduced N100 amplitude



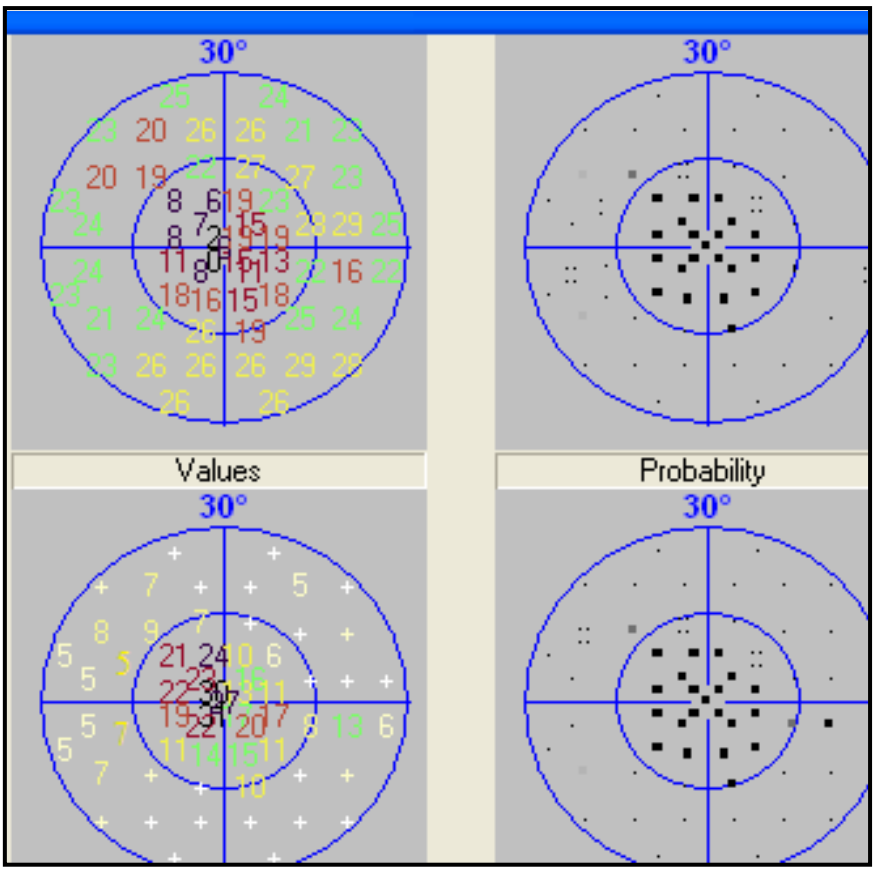
FFA of Patient No. 45 showing leakage of optic disc in optic neuritis



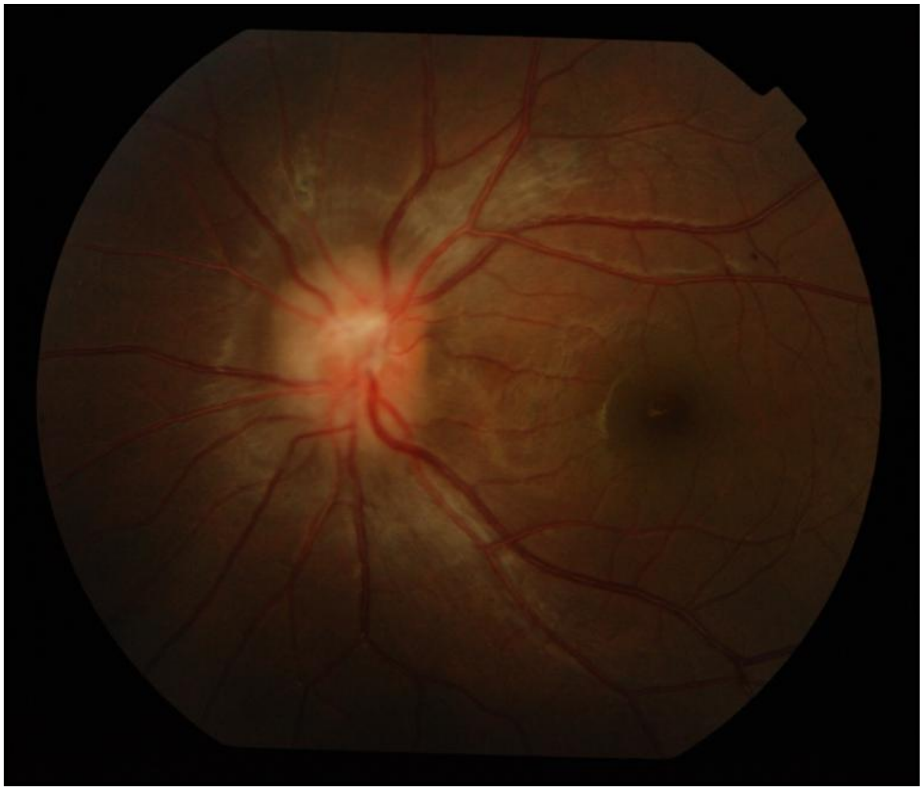
Fundus picture of patient No. 8 showing left sided optic neuritis



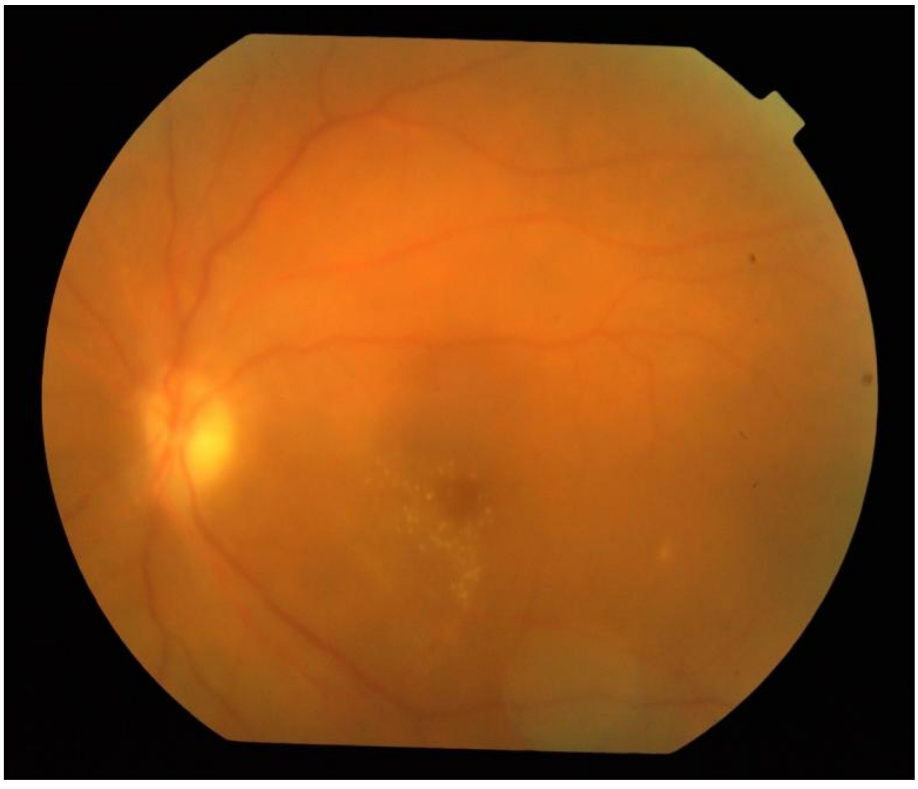
AP of patient No. 8 showing central scotoma



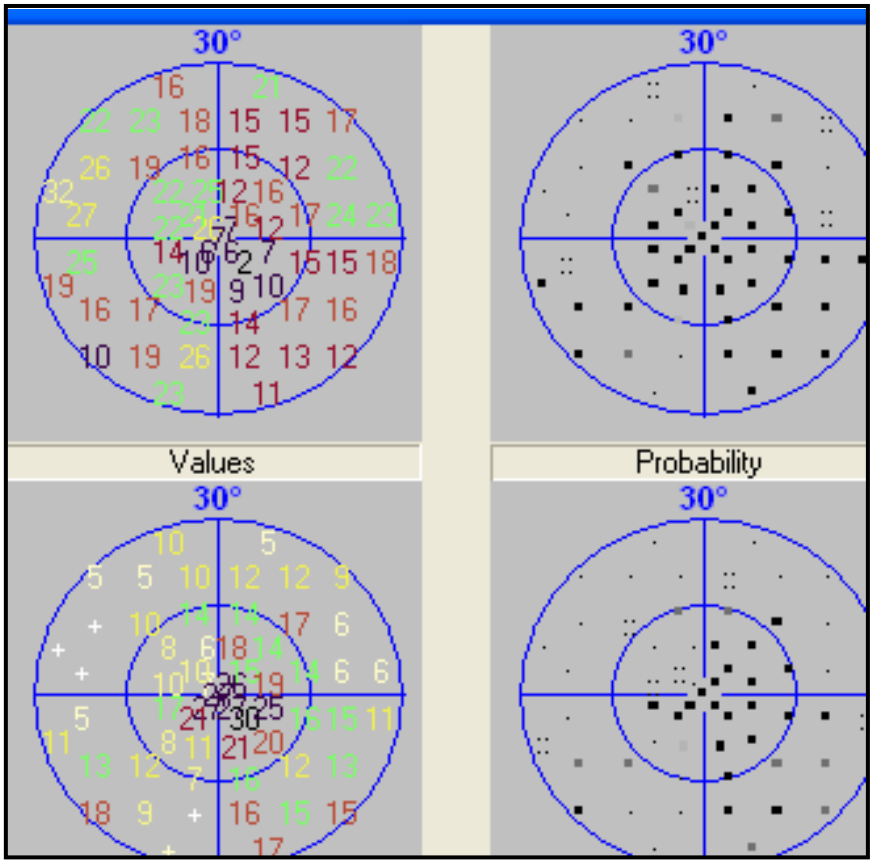
Fundus picture of Patient No. 47 showing left optic neuritis



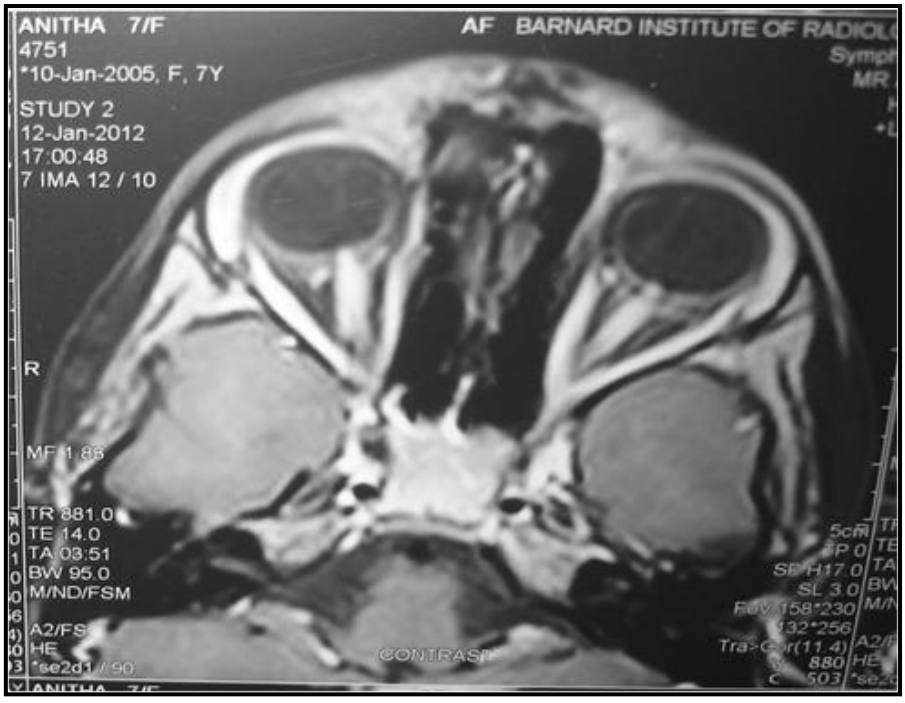
Fundus of patient No. 63 showing left optic neuritis



AP of patient 37 showing centroceacal scotoma



MRI of patient No. 33 showing Bilateral optic neuritis



DISCUSSION

All cases of optic neuritis reporting to our hospital during a period of two years were taken for study to assess the efficacy of treatment on various visual parameters including visual acuity, colour vision, pupil, fundus and fields. Analysis was done as regards to association of any specific risk factors with the final visual outcome.

In this study, totally 116 patients with 138 eyes affected with optic neuritis were studied. Among these, 50% of the patients were in the age group of 30 – 50 years which was also noted in the ONTT study. Only 6% was reported in the age group of 12 – 18 yrs.

64% of cases were females indicating a female preponderance. This female preponderance persisted through all age groups.

Defective vision was reported in 100% patients. Defective colour vision was reported in 30%. Field defects were complained in 28% of the patients. Although these complaints were co-existent with visual defect, the probable cause of this might be poor visual acuity so that colour vision and fields could not be complained of by these patients.

History of recurrence i.e. previous attack of sudden loss of vision which improved later with or without treatment was present in 10% of individuals. Approximate intervals between recurrences was 2 years in this study. History of

treatment was present in 65% of recurrent cases. But there was no difference in interval between two attack noted between the two groups (with or without treatment).

Recurrence was more common in the same eye (58%) compared to recurrence in other eye.

Considering the past history, history of fever was present in 6% of patients. Fever was more common in children. History of treatment taken for tuberculosis was present in one patient. There was no history of trauma or recent vaccination.

MRI was advised in atypical cases. It was done in seven patients in whom 4 were normal. Two patients showed signs of demyelination. One patient showed feature of optic neuritis. MRI showing demyelination in the periventricular region is a strong predictor of future development of multiple sclerosis.

Most of the cases were unilateral in 80% of patients. Bilateral cases were common in children. History of fever was more common in children which were suggestive of possibility of viral etiology for optic neuritis.

Visual acuity at the time of presentation was 6/36-6/18 in 48% of patients. Only 4% had PL vision. Colour vision was defective in 75% of patients at the time of presentation. Contrast sensitivity and fields were defective in all patients who had vision enough to be tested. Central scotoma was present in 42% of patients

and non specific defects were noted in 45% of patients. During follow up fields were tested by Bjerrum's screen. Those who had abnormal field were subjected to automated perimetry at three months all of whom showed persistent field defect. Abnormal pupillary reaction was noted in all the patients. Even after treatment pupillary abnormality persisted in 25% of patients. Retro bulbar neuritis was noted in 28% of the patients. Fundus abnormalities were resolved by three months in all patients. 8% of patients developed temporal pallor at three months of follow up indicating some residual damage to optic nerve.

After ONTT, visual acuity abnormalities resolved in 76% of patients by one month and 94% in three months. Colour vision and contrast sensitivity abnormality persisted even after three months.

ONTT hastened improvement of visual acuity in all the patients with optic neuritis.

CONCLUSION

- Females were predominantly affected than males in the ratio of 3:1.
- Most common affected patients were in the age group of 30-50 yrs.
- Recurrent cases included 10% of the patients. Recurrence was more common in the same eye.
- No specific association with any etiological factor was noted.
- Almost all patients had defective vision. Around 25% had complaints of defective colour vision, defective field of vision and 60% had ocular pain.
- Bilateral cases were around 18%. They were more common in children.
- Fever was more common in children and more commonly associated with bilateral optic neuritis.
- Visual acuity at the time of presentation was 6/60 – 6/36. In all the patients pupillary reaction was abnormal. Colour vision was abnormal in 75% of patients. Contrast sensitivity was abnormal in all patients.
- Field defect was always noted in all patients most common was central scotoma.
- Retrobulbar neuritis was noted in 28% of patients. Other patients had optic neuritis. Most commonly disc oedema was noted. Rarely haemorrhage was noted.
- After ONTT almost all patients showed improvement in vision. This improvement of vision is statistically significant.
- Colour vision recovery was noted in 46% after three months of follow up.
- Fields showed persistent field defect after three months in the patients.
- Institution of ONTT has definite role in speeding up of recovery.

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PROFORMA FOR FACTORS INFLUENCING OUTCOME

OF OPTIC NEURITIS

Name	
Age/Sex	
O.P./I.P. No.	
Date	
Address	
Contact No.	
Unit	
Diagnosis	
Complaints	

History

Sl.No.	Complaints	
1.	Loss/Blurring of Vision - Onset Duration Progression Whether increased with - exercise or hot bath - bright light	
2.	Pain - Associated with movement of eyeball Tender sore eye	

3.	Distorted Vision	
4.	Black area with in visual field	
5.	Decrease in Peripheral Vision	
6.	Defective colour vision esp red	
7.	Flashes of light	
8.	Loss of contrast sensitivity	
9.	History related to orbital inflammation - eye swelling proptosis	

Relevant History

Sl. No.	History	
1.	Fever, Head ache, Nausea, Vomiting	
2.	H/o. alcohol intake (methylated spirit)	
3	H/o. Tobacco smoking / chewing / snuffing	
4.	H/o. Tuberculosis, Syphilis	
5.	H/o. Viral infections (Herpes, measles, mumps, HBV, HIV)	
6.	H/o. Sinusitis	
7.	H/o. Diabetes mellitus	
8.	H/0. Seizures	
9.	H/o. Thyroid disease (Grave s)	
10.	H/o. Drug intake eg. Ethambutol, INH, Streptomycin, Chloramphenicol, Amiodarone	
11.	H/o. Radiation to head	
12.	H/o. Exposure to toxins eg. Lead, arsenic	

13.	H/o. Bee sting	
14.	H/o. Vaccination eg. Influenza, Hepatitis B, Measles	
15.	H/o. Similar episode (loss of vision) before	
16.	H/o. Similar episode in other family members	

EXAMINATION:

GENERAL EXAMINATION:

Physical Examination: Built

Nourishment

Anemia

Temperature

Features related to autoimmune disease

Musculoskeletal system

Skin

CNS Examination :

Higher functions –

Motor system –

Sensory system –

Cerebellar system –

Spinal cord –

Abdominal examination :

Cardiovascular system :

Respiratory system :

OCULAR EXAMINATION: SLIT LAMP EXAMINATION

	RE	LE
Visual Acuity		
Lids		
Conjunctiva		
Cornea		
AC		
Pupil	Normal / RAPD Direct Indirect	Normal / RAPD Direct Indirect
Iris		
Lens		
Intra ocular tension		
Extra ocular movements		
Pain / tenderness on mvt of eyeball		

Colour Vision RE LE

Intra Ocular Tension RE LE

Fundus Examination:

Direct Ophthalmoscopy Media

Optic disc

CD ratio

Indirect Opthalmoscopy

Retinoscopy and AR subjective

Field of Vision

	RE	LE
Manual		
AP		
Inference :		

PROVISIONAL DIAGNOSIS

INVESTIGATIONS:

Ocular Investigations :

1. FFA

2. B Scan

3. Visual Evoked Potential

General investigations :

1. Complete hemogram

    TC

    DC

    ESR

    Hb

2. VDRL

3. Mantoux test

4. Chest X Ray

5. MRI Brain with Gadolinum contrast

Management

Investigation summary

Positive Findings	Negative Findings

Treatment

Intravenous treatment			
Date	**Steroids**	**Neuro vitamins**	**Methyl Prednisolone Dose**

Treatment Summary:

Visual Acuity during treatment period

Date	Day of admission	Visual Acuity	
		RE	LE

FOLLOW UP :

Date	Visual Acuity		Field of Vision		Colour Vision		Fundus	Pupil
	RE	LE	RE	LE	RE	LE		

Master Chart

Pre Treatment Chart

Patients No : 1 -17

SlNo.	Name	Age	Sex	Complaint	Fever	Relevant H/o.	CNS examination	Visual acuity		Pupil		Fundus		Colour vision		Contrast sensitivity		Fields		ONTT	Complete hemogram	Chest X Ray	MRI Brain	VEP
								RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					
1	Visalam	29	F	a,b	-	-	N	6/6	6/36	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
2	Muthiah	33	M	a,b,c	-	a+	N	6/6	5/60	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
3	Ashik Nisha	56	F	a,b	-	-	N	6/6	6/36	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
4	Annamalai	34	M	a,b,c	-	-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
5	Gowthami	7	F	a,b	+	-	N	5/60	4/60	Ill	Ill	Ab	Ab	↓	NP	↓	NP	NP	NP	Given	Ab	N		
6	Koilmani	28	M	a	-	-	N	6/6	PL	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		
7	Kalaiarasi	33	F	a,b	-	a-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CC	N	Given	N	N		
8	Sujatha	54	F	a	-	-	N	6/6	6/24	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
9	Therasa	32	F	a,b,c	-	-	N	3/60	6/6	RAPD	N	N	N	NP	N	NP	N	NP	N	Given	N	N		
10	Imayavalli	6	F	a	-	-	N	6/36	6/6	RAPD	N	Ab	N	↓	N	↓	N	NP	N	Given	Ab	N		
11	Kameswari	52	F	a,c	-	a+	N	6/18	6/6	RAPD	N	Ab	N	↓	N	↓	N	NS	N	Given	N	N		
12	Shanthi	24	F	a,b,d	-	-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
13	Sadhurya	4	F	a	+	-	N	6/36	6/36	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NP	NP	Given	Ab	N		
14	Vignesh	36	M	a,b,c	-	-	N	6/9	6/6	RAPD	N	N	N	↓	N	↓	N	CC	N	Given	N	N		
15	Sirisha	51	F	a,c	-	e	N	6/6	6/36	N	RAPD	N	Ab	N	↓	N	↓	N	NS	Given	N	N		
16	Anjali	15	F	a,b,d	-	-	N	6/6	2/60	N	RAPD	N	N	N	NP	N	NP	N	NP	Given	N	N		
17	Jayanthi	27	F	a	-	-	N	6/24	6/18	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N	Ab	

Patients No : 18 - 35

SlNo.	Name	Age	Sex	Complaint	Fever	Relevant H/o.	CNS examination	Visual acuity		Pupil		Fundus		Colour vision		Contrast sensitivity		Fields		ONTT	Complete hemogram	Chest X Ray	MRI Brain	VEP
								RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					
18	Radha	51	F	a,b	-	-	N	6/12	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
19	Madhusudhan	5	M	a,c	+	-	N	6/18	6/6	RAPD	N	Ab	N	↓	N	↓	N	NP	N	Given	Ab	N		
20	Arockiamary	53	F	a,b,d	-	-	N	HM	6/6	RAPD	N	Ab	N	NP	N	NP	N	NP	N	Given	N	N		
21	Kanniappan	22	M	a	-	-	N	6/36	6/24	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N		
22	Munikannan	17	M	a,b,c	-	-	N	6/6	1/60	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		
23	Elumalai	42	M	a,c	-	a+	N	PL	6/6	RAPD	N	Ab	N	NP	N	NP	N	NP	N	Given	N	N		
24	Lakshmi.S	22	F	a,b	-	-	N	6/6	3/60	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		
25	Kalaivani	24	F	a	+	-	N	6/36	6/18	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NP	NP	Given	Ab	N		
26	Srijith	54	M	a,b,c	-	e	N	6/6	5/60	N	RAPD	N	Ab	N	↓	N	↓	N	CC	Given	N	N		
27	Hari	43	M	a	-	b	N	CFCF	6/6	RAPD	N	Ab	N	NP	N	NP	N	NP	N	Given	N	N		
28	Kesarvaman	25	M	a,b,c	+	-	N	6/24	6/36	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	Ab	N	N	
29	Suseela	57	F	a,b	-	e	N	6/36	6/6	RAPD	N	Ab	N	N	N	↓	N	CS	N	Given	N	N		
30	Kumar	56	M	a,c	-	-	N	6/9	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	Ab		
31	Maryammal	54	F	a,b	-	e	N	6/6	3/60	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		
32	Sivagami	35	F	a,b	-	-	N	6/6	6/24	N	RAPD	N	Ab	N	↓	N	↓	N	CC	Given	N	N		
33	Anitha	7	F	a,b,c	-	a+	N	5/60	3/60	Ill	Ill	Ab	Ab	↓	NP	↓	NP	NP	NP	Given	N	N	Ab	
34	Hemalatha	52	F	a,b	-	-	N	6/6	3/60	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		
35	Kamala	14	F	a,c	-	-	N	6/36	6/18	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	CS	Given	N	N	N	

Patients No : 36 - 53

SlNo.	Name	Age	Sex	Complaint	Fever	Relevant H/o.	CNS examination	Visual acuity		Pupil		Fundus		Colour vision		Contrast sensitivity		Fields		ONTT	Complete hemogram	Chest X Ray	MRI Brain	VEP
								RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					
36	Valliammal	37	F	a,b,c	-	a-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
37	Padma	38	F	a	-	-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CC	N	Given	N	N		
38	Mahesh	54	M	a,b,c	-	e	N	6/24	6/6	RAPD	N	Ab	N	↓	N	↓	N	NS	N	Given	N	N		
39	Manoharan	6	M	a,b	+	-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	NP	N	Given	Ab	N		
40	Govindaraj	34	M	a,b,c	-	-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		ON
41	Kothandaraman	35	M	a,b	-	-	N	6/36	6/18	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	CS	Given	N	N		
42	Perumal	52	M	a,b,c	-	-	N	6/36	6/6	RAPD	N	Ab	N	N	N	↓	N	CC	N	Given	N	N		
43	Nagammal	25	F	a,b,d	-	-	N	HM	6/6	RAPD	N	N	N	NP	N	NP	N	NP	N	Given	N	N		
44	Nalini	11	F	a,b,c	-	-	N	6/36	6/24	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NP	NP	Given	N	N		
45	Annadurai.K	48	M	a,b,d	-	e	N	6/36	6/6	RAPD	N	N	N	↓	N	↓	N	NS	N	Given	N	N		
46	Ashok Kumar	35	M	a	-	a+	N	6/12	6/6	RAPD	N	Ab	N	N	N	↓	N	NS	N	Given	N	N		
47	Arunkumar	20	M	a,b,c	-	-	N	6/6	6/60	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
48	Saravanan	37	M	a,b,d	-	-	N	6/6	3/60	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		
49	AnilBabu	37	M	a,b	-	-	N	6/6	5/60	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
50	Selvi	30	F	a,b,c,d	-	-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
51	ChadraBose	37	M	a,c	-	-	N	HM	6/6	RAPD	N	Ab	N	NP	N	↓	NP	NP	N	Given	N	N		
52	Nedunchezian	17	M	a,b,d	-	-	N	6/36	6/24	Ill	Ill	N	N	↓	↓	↓	↓	NS	NS	Given	N	N		
53	Raja	52	M	a	-	-	N	6/12	6/6	RAPD	N	Ab	N	↓	N	↓	N	NS	N	Given	N	N		ON

Patients No : 54 - 71

SlNo.	Name	Age	Sex	Complaint	Fever	Relevant H/o.	CNS examination	Visual acuity		Pupil		Fundus		Colour vision		Contrast sensitivity		Fields		ONTT	Complete hemogram	Chest X Ray	MRI Brain	VEP
								RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					
54	Geetha	56	F	a,c,d	-	-	N	6/6	1/60	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		
55	Valliamma	15	F	a,b	-	-	N	6/6	6/18	N	RAPD	N	N	N	N	N	↓	N	CC	Given	N	N		
56	Lakshmi.K	40	F	a,b,d	-	a+	N	6/6	6/60	N	RAPD	N	N	N	N	N	↓	N	NS	Given	N	N		
57	Mariammal	56	F	a,b,d	-	-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
58	Duraisamy	39	M	a	-	e	N	HM	6/6	RAPD	N	N	N	NP	N	↓	NP	NP	N	Given	N	N		
59	Iyappan	11	M	a,b	-	-	N	6/36	6/24	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NP	NP	Given	N	N		
60	Arasalingam	41	M	a	-	-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
61	Gunasekaran	39	M	a,b,d	-	-	N	6/36	6/6	RAPD	N	N	N	↓	N	↓	N	NS	N	Given	N	N		
62	Rajendran	56	M	a,b,d	-	-	N	6/18	6/6	RAPD	N	Ab	N	↓	N	↓	N	NS	N	Given	N	N		
63	Abitha	6	F	a,b	-	-	N	6/6	6/36	N	RAPD	N	Ab	N	↓	N	↓	N	NP	Given	N	N		
64	Suseela raman	57	F	a,b,d	-	-	N	6/6	5/60	N	RAPD	N	Ab	N	↓	N	↓	N	CC	Given	N	N		
65	Thanam	29	F	a,d	-	-	N	5/60	HM	Ill	Ill	N	N	↓	NP	↓	NP	NS	NP	Given	N	N		
66	Ramar	44	M	a	-	e	N	1/60	6/6	RAPD	N	Ab	N	NP	N	NP	N	NP	N	Given	N	N		ON
67	Subalakshmi	42	F	a	-	e	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
68	Rani	48	F	a	-	e	N	6/6	6/9	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
69	Annammal	45	F	a,c,d	-	a-	N	6/6	6/18	N	RAPD	N	N	N	↓	N	↓	N	NS	Given	N	N	Ab	
70	Subramaniam	29	M	a	-	-	N	6/36	6/24	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N		
71	Santhanam	46	M	a,b,c,d	-	-	N	6/6	1/60	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		

Patients No : 72 -89

SlNo.	Name	Age	Sex	Complaint	Fever	Relevant H/o.	CNS examination	Visual acuity		Pupil		Fundus		Colour vision		Contrast sensitivity		Fields		ONTT	Complete hemogram	Chest X Ray	MRI Brain	VEP
								RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					
72	Mohan	14	M	a,b	-	-	N	6/24	6/36	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N	N	
73	Kumaran	35	M	a,b	-	-	N	6/36	6/6	RAPD	N	Ab	N	↓	N	↓	N	CC	N	Given	N	N		
74	Marial	26	F	a,b	-	a+	N	PL	6/6	RAPD	N	N	N	NP	N	NP	N	NP	N	Given	N	N	N	
75	Jeevanandam	36	M	a	-	-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	NS	N	Given	N	N		
76	Tamilarasan	48	M	a,b,d	-	-	N	6/36	6/6	RAPD	N	Ab	N	N	N	↓	N	CS	N	Given	N	N		
77	Subbammal	43	F	a	-	-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
78	Sabari	51	F	a,b	-	-	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
79	Thangalaxmi	26	F	a,d	-	-	N	6/36	6/18	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N	N	
80	Rajathi	38	F	a,b,c	-	-	N	6/6	6/24	N	RAPD	N	N	N	↓	N	↓	N	CS	Given	N	N		
81	Amala	18	F	a,b	-	-	N	5/60	5/60	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N		
82	Parvathy	36	F	a,b,c	-	-	N	6/36	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
83	Chitrarani	46	F	a,b	-	-	N	6/36	6/6	RAPD	N	Ab	N	N	N	↓	N	NS	N	Given	N	N		
84	Lakshmi	42	F	a,b,c,d	-	-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
85	Uma	4	F	a	-	-	N	6/36	6/6	RAPD	N	Ab	N	N	N	↓	N	NP	N	Given	N	N		
86	Kailasam	49	F	a,b,d	-	-	N	6/6	6/12	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
87	Meena	27	F	a	-	-	N	6/36	6/18	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N		
88	Sornam	38	F	a,b,c,d	-	-	N	6/6	5/60	N	RAPD	N	N	N	↓	N	↓	N	CS	Given	N	N		
89	Sorimuthu	32	M	a	-	-	N	PL	6/6	RAPD	N	Ab	N	NP	N	NP	N	NP	N	Given	N	N		

Patients No : 90 - 107

SlNo.	Name	Age	Sex	Complaint	Fever	Relevant H/o.	CNS examination	Visual acuity		Pupil		Fundus		Colour vision		Contrast sensitivity		Fields		ONTT	Complete hemogram	Chest X Ray	MRI Brain	VEP
								RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					
90	Palaniammal	39	F	a,b	-	-	N	6/6	5/60	N	RAPD	N	N	N	↓	N	↓	N	CS	Given	N	N		
91	Vijaya	42	F	a	-	-	N	6/6	6/9	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
92	Sivam	49	M	a,b	-	-	N	6/6	6/36	N	RAPD	N	Ab	N	↓	N	↓	N	NS	Given	N	N		
93	Bargavy	43	F	a	-	e	N	6/6	6/24	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
94	Kavitha	43	F	a,b	-	-	N	6/6	5/60	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
95	Kannammal	35	F	a	-	-	N	PL	6/6	RAPD	N	N	N	NP	N	NP	N	NP	N	Given	N	N		
96	Sekar	12	M	a,b	+	-	N	6/36	6/36	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NP	NP	Given	Ab	N		
97	Baby	36	F	a,b,d	-	-	N	6/6	6/36	N	RAPD	N	N	N	↓	N	↓	N	CS	Given	N	N		
98	Jothi	37	F	a,c	-	-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
99	Rajalakshmi	24	F	a,d	-	-	N	6/18	6/36	Ill	Ill	N	N	↓	↓	↓	↓	NS	NS	Given	N	N		
100	Subarani	33	F	a	-	-	N	6/36	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
101	Kalai	42	F	a,c	-	e	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	CS	N	Given	N	N		
102	Shoba	49	F	a	-	-	N	6/6	5/60	N	RAPD	N	N	N	↓	N	↓	N	CS	Given	N	N		
103	Subashree	21	F	a,b,d	-	-	N	6/36	6/24	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NS	NS	Given	N	N		
104	Vani	45	F	a,b	-	e	N	6/6	6/36	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
105	Annadurai.D	31	M	a,b,c	-	a-	N	6/6	5/60	N	RAPD	N	N	N	↓	N	↓	N	NS	Given	N	N		
106	Nithi	42	F	**a,d**	-	-	N	6/60	6/6	RAPD	N	N	N	N	N	↓	N	NS	N	Given	N	N		
107	Suba	41	F	a,b	-	-	N	6/6	PL	N	RAPD	N	Ab	N	NP	N	NP	N	NP	Given	N	N		

Patients No : 108 - 116

SlNo.	Name	Age	Sex	Complaint	Fever	Relevant H/o.	CNS examination	Visual acuity		Pupil		Fundus		Colour vision		Contrast sensitivity		Fields		ONTT	Complete hemogram	Chest X Ray	MRI Brain	VEP
								RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE					
108	Thangam	12	F	a	-	-	N	6/36	6/18	Ill	Ill	Ab	Ab	↓	↓	↓	↓	NP	NP	Given	Ab	N		
109	Latha	47	F	a,b,d	-	e	N	6/6	6/36	N	RAPD	N	Ab	N	N	N	↓	N	NS	Given	N	N		
110	Shruti	22	F	a,b	-	-	N	6/6	6/60	N	RAPD	N	N	N	↓	N	↓	N	NS	Given	N	N		
111	Raji	37	F	a	-	e	N	5/60	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
112	Jayamaran	22	M	a,b,d	-	-	N	6/24	6/6	RAPD	N	N	N	↓	N	↓	N	NS	N	Given	N	N		
113	Revathy	34	F	a,d	-	-	N	6/6	6/9	N	RAPD	N	Ab	N	↓	N	↓	N	CS	Given	N	N		
114	Reka	28	F	a	-	-	N	6/36	6/6	RAPD	N	Ab	N	↓	N	↓	N	CS	N	Given	N	N		
115	Rajammal	35	F	a,b	-	a+	N	1/60	6/6	RAPD	N	N	N	NP	N	NP	N	NP	N	Given	N	N		
116	Meenakumari	35	F	a	-	-	N	5/60	6/6	RAPD	N	N	N	↓	N	↓	N	NS	N	Given	N	N		

Post Treatment Chart

Patients No : 1 -17

				One week								One month								Three months									
Sl No	**Name**	**Age**	**Sex**	**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Fields**	
				RE	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**	**RE**	**LE**
1	Visalam	29	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
2	Muthiah	33		6/6	6/36	N	ill	N	Ab	N	↓	6/6	6/24	N	ill	N	N	N	N	6/6	6/18	N	ill	N	N	N	N		
3	Ashik Nisha	56	F	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
4	Annamalai	34	M	6/6	6/6	N	N	Ab	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
5	Gowthami	7	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
6	Koilmani	28	M	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
7	Kalaiarasi	33	F	6/36	6/6	ill	N	N	N	N	N	6/36	6/6	ill	N	N	N	N	N	6/9	6/6	ill	N	N	N	N	N		
8	Sujatha	54	F	6/6	6/18	N	ill	N	N	N	↓	6/6	6/18	N	ill	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
9	Therasa	32	F	6/18	6/6	ill	N	N	N	N	N	6/6	6/6	ill	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
10	Imayavalli	6	F	6/24	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	N	N	6/24	6/6	ill	N	N	N	N	N		
11	Kameswari	52	F	6/12	6/6	N	ill	N	N	N	N	6/9	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
12	Shanthi	24	F	6/18	6/6	ill	ill	N	N	↓	N	6/9	6/6	ill	N	N	N	↓	N	6/9	6/6	ill	N	N	N	N	N		
13	Sadhurya	4	F	6/18	6/12	ill	ill	N	N	↓	↓	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
14	Vignesh	36	M	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
15	Sirisha	51	F	6/6	6/18	N	N	N	Ab	N	↓	6/6	6/18	N	N	N	Ab	N	↓	6/6	6/18	N	N	N	N	N	↓		
16	Anjali	15	F	6/6	6/36	N	ill	N	N	N	N	6/6	6/6	N	ill	N	N	N	N	6/6	6/6	N	ill	N	N	N	N		
17	Jayanthi	27	F	6/12	6/12	ill	ill	N	N	N	↓	6/9	6/12	ill	ill	N	N	N	N	6/9	6/12	ill	ill	N	Tp	N	N	N	NS

Patients No : 18 -38

				One week								One month								Three months									
Sl No	**Name**	**Age**	**Sex**	**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Fields**	
18	Radha	51	F	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N		
19	Madhusudhan	5	M	6/12	6/6	N	ill	N	N	↓	N	6/12	6/6	N	N	N	N	↓	N	6/12	6/6	N	N	N	N	↓	N		
20	Arockiamary	53	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
21	Kanniappan	22	M	6/18	6/12	ill	ill	N	N	↓	↓	6/9	6/12	ill	ill	N	N	N	↓	6/9	6/6	ill	ill	N	N	N	↓		
22	Munikannan	17	M	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
23	Elumalai	42	M	6/36	6/6	ill	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N		
24	Lakshmi.S	22	F	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
25	Kalaivani	24	F	6/18	6/12	N	ill	N	N	N	↓	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
26	Srijith	54	M	6/6	6/36	N	ill	N	N	N	N	6/6	6/36	N	ill	N	N	N	N	6/6	6/36	N	N	N	N	N	N		
27	Hari	43	M	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
28	Kesarvaman	25	M	6/12	6/6	N	ill	N	N	↓	↓	6/12	6/6	N	ill	N	N	↓	↓	6/12	6/6	N	ill	N	Tp	↓	↓	NS	NS
29	Suseela	57	F	6/18	6/6	ill	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
30	Kumar	56	M	6/9	6/6	ill	N	Ab	N	↓	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
31	Maryammal	54	F	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
32	Sivagami	35	F	6/6	6/12	N	ill	N	N	N	↓	6/6	6/9	N	ill	N	N	N	↓	6/6	6/9	N	ill	N	N	N	N		
33	Anitha	7	F	6/12	6/12	N	N	N	N	↓	N	6/9	6/9	N	Ill	N	N	↓	N	6/9	6/9	N	Ill	N	N	↓	N		
34	Hemalatha	52	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
35	Kamala	14	F	6/12	6/12	N	ill	N	N	↓	↓	6/6	6/12	N	ill	N	N	N	N	6/6	6/12	N	ill	N	Tp	N	N	N	CS
36	Valliammal	37	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
37	Padma	38	F	6/9	6/6	N	N	Ab	N	↓	N	6/9	6/6	N	N	Ab	N	N	N	6/9	6/6	N	N	N	N	N	N		
38	Mahesh	54	M	6/18	6/6	ill	N	N	N	N	N	6/9	6/6	ill	N	N	N	N	N	6/9	6/6	ill	N	N	N	N	N		

Patients No : 39 -59

				One week								One month								Three months									
Sl No	**Name**	**Age**	**Sex**	**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Fields**	
39	Manoharan	6	M	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
40	Govindaraj	34	M	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
41	Kothandaraman	35	M	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
42	Perumal	52	M	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	Tp	N	N	N	N	CC
43	Nagammal	25	F	6/36	6/6	ill	N	N	N	N	N	6/6	6/6	ill	N	N	N	N	N	6/6	6/6	ill	N	N	N	N	N		
44	Nalini	11	F	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓		
45	Annadurai.K	48	M	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N		
46	Ashok Kumar	35	M	6/9	6/6	ill	N	N	N	N	N	6/9	6/6	N	N	N	N	N	N	6/9	6/6	N	N	N	N	N	N		
47	Arunkumar	20	M	6/6	6/24	N	ill	N	N	N	↓	6/6	6/9	N	ill	N	N	N	↓	6/6	6/9	N	ill	N	N	N	↓		
48	Saravanan	37	M	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
49	AnilBabu	37	M	6/6	6/24	N	ill	N	N	N	↓	6/6	6/24	N	ill	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
50	Selvi	30	F	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	Tp	N	N	N	N	CS
51	ChadraBose	37	M	6/36	6/6	ill	N	N	N	↓	N	6/36	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N		
52	Nedunchezian	17	M	6/9	6/6	N	N	N	N	↓	↓	6/9	6/6	N	N	N	N	↓	↓	6/9	6/6	N	N	N	N	↓	↓		
53	Raja	52	M	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
54	Geetha	56	F	6/6	6/36	N	ill	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
55	Valliamma	15	F	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
56	Lakshmi.K	40	F	6/6	6/18	N	ill	N	N	N	N	6/6	6/9	N	ill	N	N	N	N	6/6	6/9	N	ill	N	N	N	N		
57	Mariammal	56	F	6/24	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N		
58	Duraisamy	39	M	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
59	Iyappan	11	M	6/6	6/6	N	N	Ab	Ab	↓	↓	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓		

Patients No : 60-80

				One week								One month								Three months									
Sl No	**Name**	**Age**	**Sex**	**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Fields**	
60	Arasalingam	41	M	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N		
61	Gunasekaran	39	M	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
62	Rajendran	56	M	6/6	6/6	ill	N	N	N	N	N	6/6	6/6	ill	N	N	N	N	N	6/6	6/6	ill	N	Tp	N	N	N	N	NS
63	Abitha	6	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
64	Suseela raman	57	F	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
65	Thanam	29	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
66	Ramar	44	M	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N		
67	Subalakshmi	42	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
68	Rani	48	F	6/6	6/9	N	N	N	Ab	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
69	Annammal	45	F	6/6	6/12	N	ill	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
70	Subramaniam	29	M	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	Tp	N	↓	N	NS	NS
71	Santhanam	46	M	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
72	Mohan	14	M	6/6	6/9	N	N	N	N	↓	↓	6/6	6/9	N	N	N	N	↓	↓	6/6	6/9	N	N	N	N	↓	↓		
73	Kumaran	35	M	6/12	6/6	ill	N	Ab	N	↓	N	6/12	6/6	N	N	N	N	↓	N	6/12	6/6	N	N	N	N	↓	N		
74	Marial	26	F	6/24	6/6	ill	N	N	N	N	N	6/18	6/6	ill	N	N	N	N	N	6/18	6/6	ill	N	N	N	N	N		
75	Jeevanandam	36	M	6/18	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N		
76	Tamilarasan	48	M	6/18	6/6	ill	N	N	N	N	N	6/6	6/6	ill	N	N	N	N	N	6/6	6/6	ill	N	N	N	N	N		
77	Subbammal	43	F	6/18	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N		
78	Sabari	51	F	6/6	6/6	N	N	Ab	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
79	Thangalaxmi	26	F	6/12	6/12	N	N	N	N	↓	↓	6/12	6/12	N	Ill	N	N	↓	↓	6/12	6/12	N	Ill	N	N	↓	↓		
80	Rajathi	38	F	6/6	6/12	N	ill	N	N	N	↓	6/6	6/6	N	ill	N	N	N	↓	6/6	6/6	N	ill	N	N	N	↓		

Patients No : 81-101

				One week								One month								Three months									
Sl No	Name	Age	Sex	Visual acuity		Pupil		Fundus		Colour vision		Visual acuity		Pupil		Fundus		Colour vision		Visual acuity		Pupil		Fundus		Colour vision		Fields	
81	Amala	18	F	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓		
82	Parvathy	36	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
83	Chitrarani	46	F	6/9	6/6	N	N	N	N	N	N	6/9	6/6	N	N	N	N	N	N	6/9	6/6	N	N	N	N	N	N		
84	Lakshmi	42	F	6/18	6/6	ill	N	N	N	↓	N	6/18	6/6	N	N	N	N	↓	N	6/18	6/6	N	N	Tp	N	↓	N	CS	NS
85	Uma	4	F	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
86	Kailasam	49	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
87	Meena	27	F	6/6	6/6	ill	N	N	N	↓	↓	6/6	6/6	ill	N	N	N	↓	↓	6/6	6/6	ill	N	N	N	↓	↓		
88	Sornam	38	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
89	Sorimuthu	32	M	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
90	Palaniammal	39	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
91	Vijaya	42	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
92	Sivam	49	M	6/6	6/12	N	ill	N	N	N	↓	6/6	6/6	N	ill	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
93	Bargavy	43	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
94	Kavitha	43	F	6/6	6/9	N	N	N	Ab	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
95	Kannammal	35	F	6/24	6/6	N	ill	N	N	N	N	6/9	6/6	N	N	N	N	N	N	6/9	6/6	N	N	N	N	N	N		
96	Sekar	12	M	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	N	↓		
97	Baby	36	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	N		
98	Jothi	37	F	6/12	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N		
99	Rajalakshmi	24	F	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓		
100	Subarani	33	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
101	Kalai	42	F	6/24	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N	6/6	6/6	ill	N	N	N	↓	N		

Patients No : 102-116

				One week								One month								Three months									
Sl No	**Name**	**Age**	**Sex**	**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Visual acuity**		**Pupil**		**Fundus**		**Colour vision**		**Fields**	
102	Shoba	49	F	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
103	Subashree	21	F	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓		
104	Vani	45	F	6/6	6/9	N	N	N	Ab	N	↓	6/6	6/9	N	N	N	N	N	↓	6/6	6/9	N	N	N	N	N	↓		
105	Annadurai.D	31	M	6/6	6/12	N	ill	N	N	N	↓	6/6	6/6	N	ill	N	N	N	↓	6/6	6/6	N	N	N	Tp	N	↓	N	NS
106	Nithi	42	F	6/12	6/6	ill	N	N	N	N	N	6/9	6/6	ill	N	N	N	N	N	6/9	6/6	ill	N	N	N	N	N		
107	Suba	41	F	6/6	6/24	N	ill	N	N	N	N	6/6	6/24	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
108	Thangam	12	F	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓	6/6	6/6	N	N	N	N	↓	↓		
109	Latha	47	F	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N	6/6	6/6	N	N	N	N	N	N		
110	Shruti	22	F	6/6	6/36	N	ill	N	N	N	↓	6/6	6/36	N	ill	N	N	N	↓	6/6	6/36	N	ill	N	Tp	N	N	N	NS
111	Raji	37	F	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N	6/6	6/6	N	N	N	N	↓	N		
112	Jayamaran	22	M	6/12	6/6	ill	N	N	N	↓	N	6/12	6/6	ill	N	N	N	↓	N	6/12	6/6	ill	N	N	N	↓	N		
113	Revathy	34	F	6/6	6/6	N	N	N	Ab	N	↓	6/6	6/6	N	N	N	N	N	↓	6/6	6/6	N	N	N	N	N	↓		
114	Reka	28	F	6/12	6/6	ill	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N		
115	Rajammal	35	F	6/18	6/6	ill	N	N	N	↓	N	6/18	6/6	ill	N	N	N	↓	N	6/18	6/6	N	N	Tp	N	↓	N	N	CS
116	Meenakumari	35	F	6/12	6/6	ill	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N	6/9	6/6	N	N	N	N	↓	N		

KEYNOTE TO MASTER CHART

Pupil

Ill - Illsustained

RAPD-Relative Afferent Pupillary Defect

Colour Vision

N - normal

↓- Red desaturation

Complaint

a - Defective vision

b - Pain in the eye

c - Defective colour vision

d-Defective field of vision

Relevant H/o.

Recurrence - present a treatment taken + not taken -

Drug intake - b

Recent vaccination - c

Trauma H/o. - d

Diabetes - e

H/o. Similar episode in other family members - f

Fundus

Normal - N

Abnormal – AbN

Complete hemogram

Normal - N

Abnormal - Ab - indicative of infection

Chest X Ray

Normal - N

Abnormal - Ab - indicative of tuberculosis

MRI Brain

Normal - N

Abnormal - Ab -indicative of Multiple sclerosis

Not done - ND

ON- s/o optic neuritis

NP - NOT POSSIBLE due to poor vision

Field

Normal N

Central scotoma CS

Centrocecal scotoma cc

Non specific – NS

Not done - ND

ONTT - optic neuritis treatment trial